DETERMINANTS OF CHILDREN'S HEALTH

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Michael Grossman, Ph.D., Principal Investigator

Douglas Coate, Ph.D., Research Associate
Linda N. Edwards, Ph.D., Research Associate
Robert A. Shakotko, Ph.D., Research Economist
Dov Chernichovsky, Ph.D., Visiting Scholar

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National Bureau of Economic Research
15-19 West 4th Street, 8th Floor
Washington Square
New York, New York 10012
(212)598-3321/3352

ABSTRACT

The purpose of this research is to investigate empirically the determinants of children's health with particular reference to home and local environmental variables such as family income, parents' schooling, preventive medical care, and health manpower availability. Wherever possible, children's health is studied in the context of the nature-nurture controversy. The findings indicate that family characteristics (especially mother's schooling) do have significant impacts on children's health and that preventive care is an important vehicle for this impact in the case of dental health but not in the case of physical health. Similarly, the greater availability of dentists has a positive impact on dental health, but greater availability of pediatricians does not alter the physical health measures.

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Michael Grossman and Linda Edwards conducted the research in Chapters 2, 3, and 4 of this report. Grossman, Edwards, and Robert Shakotko are responsible for the research in Chapter 5. Douglas Coate and Dov Chernichovsky conducted the research in Chapters 6 and 7.

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Chapter 1

INTRODUCTION

The purpose of this research is to investigate empirically the determinants of children's health with particular reference to home and local environmental variables. Home environmental factors include basic family background variables such as family income, parents' schooling, family size, and mother's labor force status. They also include mechanisms via which background variables operate such as preventive medical care and nutrition. Local environmental factors include health manpower availability, public provision of fluoridated water, climate, and air pollution.

Wherever possible, we study children's health in the context of the nature-nurture controversy. Despite the existence of a massive (but inconclusive) literature on the relative importance of heredity (nature) and the home and school environment (nurture) in the determination of cognitive development, the corresponding issue has not been directly addressed by researchers in child and adolescent health. This is partly because much of the health research is limited either to poverty or to minority populations and partly because researchers who use representative samples do not adopt the multivariate context necessary for distinguishing between genetic and environmental influences. Our research uses multivariate statistical techniques to provide some evidence of the degree to which nurture--that is, the family and local environment, acts in determining the health levels of representative samples of children and adolescents.

In Chapter 2 of this report, we examine the relationship between a number of family characteristics and the health of white children aged 6 through 11 years in the period 1963 through 1965. The data set employed is Cycle II of the U.S. Health Examination Survey (HES II). Our aim in Chapter 2 is to paint a complete picture of the health of this childhood cohort. Therefore, a variety of health measures from physical examinations by pediatricians and dentists and from interviews with parents and school officials are analyzed. In Chapter 3, we explore race and income differences in the health of children in the Cycle II data set. We compare such differences in mid-childhood to race and income differences in infant mortality and birth weight.

In Chapter 4 we investigate the health of white adolescents aged 12 through 17 years in the 1966-70 period. The data set employed is Cycle III of the Health Examination Survey. Our research in this chapter focuses on the nature-nurture controversy by examining in detail the impacts of four important components of the home and local environment: family background, preventive medical care, health manpower specific to the youth's county of residence (the number of pediatricians per capita and the number of dentists per capita), and information on the presence of controlled or natural fluorides in the water supply system that services the youth's community. Four specific health indicators are studied in Chapter 4: oral health, obesity, anemia, and corrected distance vision. These indicators are chosen because they represent health problems that are capable of being affected by family decisions concerning diet and other forms of at-home health care, as well as by pediatric and dental care.

Two types of relationships are estimated in Chapter 4: a health production function and a derived demand function for preventive care. The resulting estimates permit us to answer the following questions. What is the size of the home environmental effect on adolescent oral and physical (obesity, anemia, corrected distance vision) health outcomes? How important is the home environment as a determinant of the demand for preventive dental and pediatric care? How large are the effects of dentists, preventive dental care, and fluoridation on oral health outcomes: How large are the effects of pediatricians and preventive pediatric care on physical health outcomes? In addressing the last rwo questions, we recognize explicitly the common-sense proposition that an increase in a community's physician or dental manpower will not increase health outcomes unless it encourages more utilization of medical care services. Note that we could not study relationships among preventive medical care, health manpower, and health outcomes in Cycle II of the HES because there are no measures of preventive care in Cycle II.

In Chapter 5 of this report, we shift from cross-sectional to panel data to shed further light on the pure environmental contribution of family background in general and parents' schooling and family income in particular to the health of adolescents. We also examine whether health causes cognitive development, cognitive development causes health, or both cause each other. The sample consists of the adolescents in Cycle III of the HES who were also examined in Cycle II when they were children. This panel data set contains measures of

health and cognitive development in adolescence, corresponding measures in childhood, and family background variables.

To study the dynamics of health and cognitive development and to deal with the problem of simultaneity, we investigate the causal priorness of these measures in Chapter 5. Specifically, we assume that the processes that generate them are Markov and can be estimated by a simple first-order ARMA model. We show that, if the genetic impact on these variables is restricted to the determination of initial conditions, then the estimates of the time paths will be free of genetics-bias and will reflect the true environmental effects of family background variables. Empirically, we estimate two multivariate equations: one relating adolescent health to childhood health, childhood cognitive development, and family background; and a second relating adolescent cognitive development to childhood cognitive development, childhood health, and family background. In the health equation, the coefficient of family background gives an estimate of the environmental effect that controls for the initial or inherited level of health. The coefficient of childhood cognitive development indicates whether the latter variable causes adolescent health. The adolescent cognitive development equation can be interpreted in a similar manner. In particular, the coefficient of childhood health measures the significance and size of the causal relationship from health to cognitive development.

In Chapters 6 and 7, we investigate relationships among children's growth, diet, and family background. The growth measures are height, weight, and head circumference. The diet measures are intakes of calories and proteins. The research in Chapter 6 is based on infants

and young children between the ages of zero and 36 months in the 1968-70 period who were members of the Ten State Nutrition Survey (TSNS). The research in Chapter 7 is based on children aged 1 through 5 in the 1971-75 period who were members of Cycle I of the Health and Nutrition Examination Survey (HANES I).

Three types of empirical results are presented in Chapters 6 and 7. These are (1) descriptive statistics of variables in the TSNS and in HANES I that are important in an analysis of children's diet and growth; (2) ordinary least squares multiple regressions in which diet or growth are dependent variables and exogenous child and family characteristics are independent variables; and (3) two-stage least squares estimates of a structural model in which diet and growth are determined simultaneously.

Chapter 2

CHILDREN'S HEALTH AND THE FAMILY

I. Introduction

Children's health care has been and continues to be provided primarily within the family. This is in marked contrast to the provision of children's education which for the past 100 years has been considered a legitimate concern of government [see Landes and Solmon 1972]. While no one believes that the government can replace the family in providing education for children, state governments do determine how many years children must attend school, how many days per year they must attend, and, to a large extent, the content of that schooling. Further, recent rulings of the Supreme Court stipulate that all children are entitled to schooling of equal quality. Only recently has attention turned to the role of the preschool years, and consequently, the family, in determining the intellectual development of children.[1]

In the case of children's health, it has been widely recognized that it is the family rather than the public or medical care sectors that plays the fundamental role. For example, a recent Carnegie Council on Children report says "Doctors do not provide the bulk of health care for children; families do" [Keniston 1977, p. 179]. With the exception of immunization, which clearly has important externalities, there has been no form of compulsory health care for children,[2] and with the advent of relatively simple and effective treatments for important childhood diseases of the past (influenza, pneumonia, and tuberculosis), one might even imagine that the doctor's role is declining. Indeed, the expanding interest of pediatricians into the area of the "new morbidity"--"learning difficulties and school problems, behavioral disturbances, allergies, speech difficulties, visual

problems, and the problems of adolescents in coping and adjusting"--is a response to the decline in importance of the traditional health problems of children.[3]

While the overall importance of the family in providing health care for children is widely acknowledged, information about the nature of the association between various family characteristics and the health status of children is relatively scarce. Much of the literature concentrates on two infant health measures--infant mortality and birth weight--and documents that infants from both black families and low income families experience a higher incidence of mortality and low birth weight [Keniston 1977, p. 156]. When the health of children who survive the first year of life is the subject of study, much less is known; and again, existing studies focus primarily on health differences associated with income and race.[4] For example, there is evidence that both black families and low income families evaluate their children's health as poorer than do white or high income families. Similarly, children from black or low income families are reported to exhibit a higher incidence of "significant abnormalities" on a physical exam.[5] The extent to which these documented differences are the result of income and/or race alone, as opposed to correlated factors like parental education, family size, mother's work status, and place of residence, has hardly been studied. In order to formulate sensible and effective programs to improve the health of children, we need a much better understanding of how these and other family characteristics work in producing healthy children. Our study is a step in this direction.

More specifically, the objective of this study is to examine the relationship between a number of family characteristics and the health of children aged 6 to 11 years residing in those families. This multivariate

analysis is carried out within the framework of an economic model of the family. Such a framework explicitly recognizes not only the family's function as health care provider, but also that it is faced with resource constraints and that some of its objectives for its children may conflict with maximizing their health level.

The data set used, Cycle II of the Health Examination Survey, is an exceptional source of information about a national sample of 7,119 noninstitutionalized children aged 6 to 11 years in the 1963-65 period.[6] The data comprise complete medical and developmental histories of each child provided by the parent, information on family socioeconomic characteristics, birth certificate information, and a school report with data on school performance and classroom behavior provided by teachers or other school officials. Most important, there are objective measures of health from detailed physical examinations. The physical examinations (as well as associated psychological and achievement tests) were administered by the Public Health Service. There is little direct information about the medical care received by these children, but some attempt will be made to control for variations in the availability of local medical care.

The amount of health information for the children in the Cycle II sample is prodigious. To illustrate the exact nature of this information and to provide a description of the overall health of the children in the sample, selected summary data are presented in Table I. Panel A indicates that almost 95 percent of parents rate their child's health as good or very good. At the same time, however, 19 percent of these parents consider their child's present health to be a problem. The Public Health Service physicians affirm the latter assessment in the sense that they find that 11.2 percent of the children had at least one "significant abnormality" (see Panel B). An

TABLE I

Summary of Selected Health Information from Cycle II of the Health Examination Survey[a]

Panel A. Parent's Assessment of Children's Current Health

	Percent of Children Whose Parent Rates Present Health as:				Percent of Children Whose Parent Considers Present Health a Problem
	Very Good	Good	Fair	Poor	
Both sexes	51.8	42.9	4.9	.4	19.0
Boys	51.6	43.2	4.8	.4	19.0
Girls	51.8	42.7	5.1	.4	19.0

Panel B. Physician's Findings on Survey Examination

	Percent of Children with Finding of		Distribution of Those with Significant Abnormality by Type of Abnormality (Percent)				
	Otitis Media	Significant Abnormality	Cardio-vascular	Injury Residual	Neuro-muscular Joint	Other Congenital	Other Major Diseases
Both sexes	1.6	11.2	22.9	13.7	31.8	23.6	28.6
Boys	1.8	12.2	21.8	13.5	32.9	22.6	28.9
Girls	1.4	10.2	24.4	14.1	30.4	24.7	28.1

(continued on next page)

TABLE I (concluded)

Panel C. Medical History as Reported by Parent

Medical History Item	Both Sexes	Boys	Girls
	(percent of children with history of indicated condition)		
Accidents			
Broken bones	7.8	8.5	7.0
Knocked unconscious	3.4	4.0	2.8
Scars from burns	4.5	4.4	4.7
Other accidents	4.2	4.7	3.7
Allergies and Related Conditions			
Asthma	5.3	6.5	4.0
Hay fever	4.6	5.5	3.6
Other allergies	11.4	12.2	10.7
Kidney condition	3.9	2.6	5.1
Heart condition	3.7	4.2	3.1
Sensory-Neurological Conditions			
Convulsions or fits	3.3	3.5	3.1
Eye trouble	14.0	12.7	15.3
Trouble hearing	4.3	4.8	3.7
Earaches	26.8	24.8	28.8
Running ears	11.9	12.2	11.6
Problem talking	8.4	10.0	6.8
Trouble walking	2.3	2.5	2.1
Arm or leg limitation	1.3	1.3	1.2
Operations	30.8	35.3	26.1
Hospitalized more than 1 day	26.8	30.0	23.6
Exercise Restricted			
Ever	5.4	5.6	5.2
Now	1.5	1.4	1.6
Taking medicine regularly	4.1	4.0	4.2

[a]Source: NCHS (11)
Panel A - Table 1
Panel B - Tables 1 and 3
Panel C - Table 4

indication of the types of problems that may be bothering parents is given by the incidence of various items in the medical history (Panel C). The total picture, then, is of a cohort whose overall health is good but who are nevertheless disturbed by particular health problems.

The chapter proceeds as follows. Section II briefly describes the economic model of the family used to generate the relationships to be estimated. Section III discusses the nature of the estimated relationships and defines the variable measures. The estimates are presented and discussed in Section IV. The final sections highlight and interpret the statistical findings of the study.

II. The Economist's View of Children and Children's Health

An important focal point of recent economic models of fertility [Becker and Lewis 1973; Willis 1973; Ben Porath and Welch 1976 is that children are not homogeneous. In particular, these models distinguish between two aspects of children that enter the parents' consumption (or investment) portfolio--the number of children and the "quality" of each child. By quality of the child is meant those characteristics of the child which generate utility (or disutility) for the parents: his health, sex, wealth, social adjustment, intellectual development, sense of humor, etc. Therefore, when parents choose their optimal family composition, they choose not only how many children they will have, but also what portion of the family's resources will be devoted to each child.[7] This choice is made in the usual way: parents choose the number and quality of children, as well as of other consumption goods, so as to maximize their utility subject to the constraints imposed by their wealth (their potential earned income and their nonearned income) and the various prices they face. In the case of children, there is a further constraint in the form of children's genetic endowments which in

part determine their quality. Genetic endowments act as a constraint because they are, for the most part, outside of the family's control.[8]

The prices of children and of the various components of their quality are determined by recognizing that children are produced within the home using goods bought in the market and the time of family members. The cost of producing a child of a given quality depends on the prices of the purchased inputs--parents' time, medical care, food, toys, lodging, etc.--and on the efficiency with which these inputs are used. The marginal cost of a child, then, depends on the quality chosen and on the cost of one unit of that amount of quality. Similarly, the cost of an increment in quality depends on how many children will receive this increment (i.e. family size) and on the cost of an incremental unit of quality.[9] To take the case of the aspect of child quality focused on in this paper, children's health, the cost (price) of increasing the average health level of all children in the family depends on the number of children in the family and on the costs of medical care, nutrition, the parents' or other caretakers' time, and any other purchased inputs used to improve children's health. In addition, to the extent that there are systematic differences in the ability of families to produce children's health with given inputs, these differences in efficiency are also relevant. For example, more educated parents are more likely to be able to follow doctors instructions, to have general information about nutrition, and to be willing and able to acquire medical information from published materials. Consequently, one would expect more educated parents to be more efficient at producing healthy children.

Given these considerations, the following factors are expected to influence children's health levels: the child's exogenous (genetic) health endowment, family wealth, parents' wage rates, family size, parents'

educational attainment and other measures of their efficiency in household production, costs and availability of medical care and of other market health inputs (vitamins, sanitation, etc.), the prices of inputs used to produce other aspects of child quality, and the prices of other forms of parents' consumption. The relationship between the child's ultimate health and these factors may be termed a demand function for the output health.[10] In this demand function a positive association between children's health and family wealth is predicted (assuming that child health is a normal good). Similarly, a positive association is expected between both parents' education and children's endowed health status and children's ultimate health status.[11] Negative associations would be anticipated between all of the prices of health inputs and children's health, and between family size and children's health. Parents' wage rates may have negative or positive effects on children's health levels, depending on whether the household production of children's health is more or less time intensive than the production of other aspects of child quality and/or other types of parents' consumption commodities. Finally, to the extent that there is substitutability between the various aspects of child quality, the prices of inputs into the non-health aspects of child quality (say, music lessons) might be positively associated with children's health.[12]

These predictions concerning the effects of various family characteristics and market prices do not necessarily apply when some realistic twists are incorporated into the model. An important instance is the introduction of joint production between various aspects of child quality and/or between child quality and other consumption commodities. Such joint production can make both wealth effects and input price effects ambiguous. To take a simple example, both athletic development and health may be regarded as

aspects of children's quality. If athletic development has a high income elasticity and also has some negative health effects, a negative relationship between income or wealth and certain health measures may be observed. Indeed, the incidence of broken bones is greater in high income families than it is in low income families [NCHS 1973, Table 14]. Alternatively, suppose that parents' time in child care yields direct utility rather than generating utility only through its effect on children's quality. In this case, parents may appear to choose inefficient modes of producing child quality, modes that use "too much" time relative to its cost.

The basic model outlined above is also modified when one takes account of the fact that the human capital dimensions of child quality are by necessity embodied in the child. Because of this embodiment, for some types of human capital there are natural minimum and maximum states that cannot be reduced or exceeded. In the case of children's health, increased expenditures on health inputs or increased production efficiency cannot continually increase the child's health. For this reason, one would not predict constant absolute effects of the various determinants of children's health but rather that these effects attenuate in the region of minimum and maximum health levels.

Recent models of intergenerational transfers utilize this embodiment insight in a somewhat different way. In these models investments in children's human capital are assumed to be subject to decreasing returns in terms of the future earnings of the child. This assumption, along with the assumption that parents measure a child's quality by his expected lifetime wealth, generates additional predictions concerning the effects of the set of variables discussed earlier on various components of children's quality.[13] In particular, this type of model yields a distinction

between two types of families: those for whom the optimal quantity-quality calculation involves making a financial transfer to their children and those for whom such a financial transfer is not optimal. These two types of families differ systematically with respect to the effects of the various explanatory variables on the human capital dimensions of child quality. The strongest prediction is that the wealth effect on the child's human capital (health, in our case) will be zero in families which plan to make financial transfers to the children, but positive in families who do not plan such a transfer [see Edwards and Grossman 1977].

What is striking about this review of the economic models is that although they differ in many respects, they all designate the same set of factors as determinants of children's health--parents' wealth, wage rates, and education, family size, other input prices, etc. In addition, they provide ready structures within which to interpret empirical findings. Thus, the greater incidence of broken bones among children from high income families is seen not as an anomalous finding but rather as a result of conflicting family objectives concerning various aspects of child quality. Or, a finding that for high income families increases in wealth are not associated with increased children's health is not viewed as evidence that wealthy parents do not care about their children but rather as a result of the fact that wealthy parents have already made the optimal health investment for their children. In the sections to follow, both the choice of variables to be investigated and the interpretation of the results of that investigation are guided by the general framework outlined here.

III. Variables and Relationships to be Estimated

A. Measurement of Child Health

The issue of defining and measuring children's health is very much an unresolved one, even among professionals in the area of public health.[14] The economist's approach is to define health as a form of human capital which determines the amount of time available for consumption and for work in the home and labor market.[15] (Individuals may also derive disutility, or even utility, from the state of being ill.) With this type of definition, an appropriate measure of health status over some time period would be the proportion of potential time that is actually available for the usual consumption, maintenance, and work activities. Similarly, the complementary measure of ill health would be the proportion of potential available time lost due to inability to function or to imperfect functioning. While such disability may seem to be relatively easy to measure when dealing with adults who are members of the labor force (a good approximation is days lost from work because of illness), it is not easy to measure for other adults. Moreover, even a measure of days lost from work might not capture losses in consumption time. Therefore, in economists' studies of adults' health, both the incidence of particular physical conditions and the individual's own assessment of his health status have been used as supplementary health measures [Grossman 1972].

We use the same type of restricted, morbidity-oriented, definition of children's health--focusing on the child's physical health rather than his overall well being--and similar types of measures--disability, physical conditions, and parental assessment of health status. The use, however, of disability measures (time lost from usual activities) and of the incidence of certain physical conditions is somewhat less appropriate for children

than for adults.[16] This is because there is a natural sequence of childhood diseases and acute conditions which prevent children from carrying out their normal activities, but which do not reflect the child's health capital or the child's future prospects for life preservation and normal functioning. A useful distinction to make here is between "permanent" health, which is what we mean by health capital, and "transitory" health, those short-run deviations from one's normal state of health caused by the acute conditions of childhood.[17] It is the child's "permanent" health status that we wish to study, and we attempt to use those health measures which will be good predictors of that "permanent" health status.[18]

In some situations a single overall index of "permanent" health might be desired--to describe parsimoniously the health status of a population, or to allocate public funds. Health, however, clearly is a multidimensional concept. Analysis of a set of components rather than a single index allows us to detect differences in the effects of family background variables on the various dimensions of health. Such analysis also avoids an essentially arbitrary weighting of the various dimensions of health implied by a health index.[19]

The actual choice of components of children's health status to be examined is controlled by the Cycle II data set and guided by the child health literature,[20] as well as by discussions with public health pediatricians.[21] The measures are listed and described below.

1. The parent's assessment of the child's overall current health, represented by PFGHEALTH. PFGHEALTH is a dichotomous variable indicating whether the parent views the child's health as poor, fair, or good (as opposed to very good).

2. Current height and weight, represented by IHEIGHT and IWEIGHT. These are standard indicators of children's nutritional status [for example, National Center for Health Statistics 1970b and 1975; Seoane and Latham 1971, and good nutrition is an obvious and natural vehicle for maintaining children's health. Height is a better summary measure of the lifetime nutritional status of the child, while weight conveys information primarily about his or her current nutritional status. Since it is well known that physical growth rates differ by age and sex, our height variable is computed as the difference between the child's actual height and the mean height for his or her age-sex group divided by the standard deviation of height for that age-sex group. Our weight measure is computed in a similar manner.[22]

3. The child's hearing acuity, represented by IHEAR. IHEAR is a dichotomous variable indicating whether the child has abnormal hearing. A child is defined to have abnormal hearing if the average threshold decibel reading in his best ear over the range of 500, 1,000, and 2,000 cycles per second (c.p.s.) is greater than 15. 500, 1,000, and 2,000 c.p.s. are the frequencies that occur most frequently in normal speech. A threshold of 15 or more decibels above audiometric zero at these frequencies is classified as corresponding to "significant difficulty with faint speech" by the Committee on Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology [NCHS 1970a].

4. The child's visual acuity, represented by the dichotomous variable ABVIS. ABVIS indicates if the child has abnormal distance vision. All children were examined without their eyeglasses; their uncorrected binocular distance vision is defined as abnormal if it is worse than 20/30 [NCHS 1972].[23]

5. The child's blood pressure, represented by HDBP. HDBP is a dummy variable which indicates if the child's diastolic blood pressure exceeds the 95th percentile for his or her age and sex.

6. Whether or not the child has hayfever or other allergies, represented by the dummy variable ALLEG.

7. An assessment of the child's level of tension, represented by the dummy variable TENS. TENS characterizes children who are rated by their parents as "high strung" or "moderately tense." Both the tension and allergy variables may be regarded as measures of the "new morbidity."

8. The presence of one or more "significant acquired abnormalities" on physical examination of the child, represented by the dummy variable ACABN. These abnormalities include heart disease; neurological, muscular, or joint conditions; and other major diseases.[24]

9. The child's periodontal index, represented by APERI. APERI is a good overall indicator of oral health as well as a positive correlate of nutrition [Russell 1956]. Due to the significant age and sex trends in this variable, it is standardized by age and sex in the same manner as height and weight. Higher values of APERI denote poorer values of oral health.[25]

10. Excessive absence from school for health reasons during the past six months, represented by the dichotomous variable SCHABS. This variable is taken from information provided by the child's school.[26]

Precise definitions of the above health measures appear in Table II, along with their sample means and a notation concerning the source of each variable (medical history, physician's exam, birth certificate, or school form).

TABLE II
Definitions of Health Measures

Variable Name	Mean in Sample[a]	Definition	Source[b]
PFGHEALTH	.451	Dummy variable that equals one if parental assessment of child's health is poor, fair, or good and zero if assessment is very good	1
IHEIGHT	.071[c]	Height, standardized by the mean and standard deviation of one-year age-sex cohorts	3
IWEIGHT	.042[c]	Weight, standardized by the mean and standard deviation of one-year age-sex cohorts	3
IHEAR	.006	Dummy variable that equals one if hearing is abnormal	3
ABVIS	.116	Dummy variable that equals one if uncorrected binocular distance vision is abnormal	3
IDBP	.054	Dummy variable that equals one if the child's diastolic blood pressure is above the 95th percentile for his age and sex	3
ALLEG	.156	Dummy variable that equals one if the child has had hayfever or any other kind of allergy	1
TENS	.476	Dummy variable that equals one if the parent rates the child as high strung or moderately tense	1
ACABN	.037	Dummy variable that equals one if the physician finds a "significant" acquired abnormality in examining the child (other than an abnormality resulting from an accident or injury)	3
APERI	-.034[c]	Periodontal index, standardized by the mean and standard deviation of one-year age-sex cohorts	3
SCHABS	.045[d]	Dummy variable that equals one if child has been excessively absent from school for health reasons during the past six months	4

(footnotes on next page)

Footnotes to TABLE II

[a]These means are computed for the "basic" sample of 4,196 white children described in Section IV.

[b]The sources are 1 = medical history form completed by parent, 2 = birth certificate, 3 = physical examination, 4 = school form completed by teacher or other school official.

[c]The mean of this variable is not zero because standardization was done using the entire Cycle II sample rather than the subsample reported on in this paper.

[d]The mean of this variable is computed for a subsample of 3,812 for whom the school report is not missing.

B. Measurement of Explanatory Variables

The theoretical variables needed for investigating children's health were listed earlier: family wealth, parents' wage rates, parents' educational attainment (or other measures of their efficiency in household production), the child's health endowment, the costs and availability of medical care and other market health inputs, the prices of other inputs used to produce child quality, the prices of other forms of parents' consumption, and family size.

Not surprisingly, the actual measures available in the Cycle II survey correspond only roughly to the above theoretical variables. We discuss these measures and indicate how they relate to the theoretical variables below. A complete list of the measures, their precise definitions, and their sample means and standard deviations appear in Table III.

Family wealth and the father's wage rate are both represented by the family income measure. Two income variables are used (FINC, HFINC) in order to allow for the possibility that the relationship between family wealth and children's health differs for high and low income families.[27]

The mother's wage rate is not directly available in the survey. We attempt to control for variations in her wage rate with three variables: her educational attainment (MEDUCAT) and two measures of her current work status (MWORKFT, MWORKPT). More educated women are more likely to have higher opportunity costs, as are women who are currently in the labor force.

As efficiency (or taste) measures we use mother's and father's educational attainment (MEDUCAT, FEDUCAT) and a dummy variable identifying

TABLE III
Explanatory Variables

Variable Name	Sample Mean[a]	Sample Standard Deviation[a]	Definition[b]
FINC	7.502	4.405	Continuous family income computed by assigning midpoints to the following closed income intervals, $250 to the lowest interval, and $20,000 to the highest interval. The closed income classes are: $500 - $999 $1,000 - $1,999 $2,000 - $2,999 $3,000 - $3,999 $4,000 - $4,999 $5,000 - $6,999 $7,000 - $9,999 $10,000 - $14,999
HFINC	5.038	6.138	Same as FINC for income of $7,000 and over; equals zero for incomes below $7,000
MWORKPT MWORKFT	.132 .158	.339 .365	Dummy variables that equal one if the mother works part-time or full-time, respectively
MEDUCAT	11.095	2.808	Years of formal schooling completed by mother
FEDUCAT[c]	11.220	3.461	Years of formal schooling completed by father
FLANG	.105	.307	Dummy variable that equals one when a foreign language is spoken in the home
LIGHT1	.013	.112	Dummy variable that equals one if child's birth weight was under 2,000 grams (under 4.4 pounds)
LIGHT2	.043	.202	Dummy variable that equals one if child's birth weight was equal to or greater than 2,000 grams but under 2,500 grams (under 5.5 pounds)
CABN	.050	.219	Dummy variable that equals one if the physician finds a "significant" congenital abnormality in examining the child

TABLE III (concluded)

Variable Name	Sample Mean[a]	Sample Standard Deviation[a]	Definition[b]
MALE	.513	.500	Dummy variable that equals one if child is male
LMAG	.073	.261	Dummy variable that equals one if the mother was less than 20 years old at birth of child
HMAG	.105	.307	Dummy variable that equals one if the mother was more than 35 years old at birth of child
FIRST	.288	.453	Dummy variable that equals one if child is the first born in the family
TWIN	.023	.149	Dummy variable that equals one if child is a twin
NEAST	.236	.425	Dummy variables that equal one if child lives in Northeast, Midwest, or South, respectively; omitted class is residence in West
MWEST	.322	.467	
SOUTH	.181	.385	
URB1	.199	.400	Dummy variables that equal one if child lives in an urban area with a population of 3 million or more (URB1); in an area with a population between 1 million and 3 million (URB2); in an urban area with a population less than 1 million (URB3); or in a non-rural and non-urbanized area (NURB); omitted class is residence in a rural area
URB2	.120	.325	
URB3	.181	.385	
NURB	.148	.355	
DENT12	.169	.375	Dummy variable that equals one if child has been to a dentist sometime in his life but not within the last twelve months
DENTIST3	.179	.383	Dummy variable that equals one if child has never been to a dentist
LESS20	3.635	1.676	Number of persons in the household 20 years of age or less
NOFATH	.071	.257	Dummy variable that equals one if child lives with mother only

Footnotes to TABLE III

[a]These means and standard deviations are computed for the "basic" sample of 4,196 children described in Section IV.

[b]All of these variables except LIGHT1, LIGHT2, and CABN are taken from the medical history form. LIGHT1 and LIGHT2 come from the child's birth certificate, and CABN, from the physical examination.

[c]For children who were not currently living with their father, father's education was coded at the mean of the sample for which father's education was reported.

mothers who were under 20 years-old at the time of the child's birth (LMAG). Young mothers are notoriously less efficient at contracepting and may be similarly less efficient in producing healthy children. A supplementary efficiency (or taste) measure is a variable indicating whether a foreign language is spoken in the home (FLANG). Foreign born families are likely to exhibit differences in tastes and/or household production efficiency.

The child's endowed health status is represented by a set of variables relating to his early health. They are dummy variables identifying children of low birth weight (LIGHT1, LIGHT2), children with congenital abnormalities (CABN), the child's sex (MALE), and children whose mothers were over 35 years-old at the time of the child's birth (HMAG). Low birth weight is a typical indicator of a less healthy birth outcome.[28] The rationale for including a variable identifying older mothers is that older mothers are more likely to bear children with health defects. The child's sex is included because of the well documented higher incidence rates of certain health problems among males at young ages [for example, NCHS 1973].

In addition to these health endowment measures, we also control for several characteristics of the child which are not necessarily health related but may cause him to receive better or worse treatment within the family simply by virtue of his luck in possessing these characteristics. They are his birth-order (FIRST), whether or not he is a twin (TWIN), as well as his sex (MALE). First born children (or non-twins) will have greater access to individual parental attention because they arrived in the family first (or they arrived alone). Similarly, male children may receive larger investments than female children if males are preferred [see Ben Porath and Welch 1976].

Direct information about the prices of inputs in the health production function and in the other household production functions is not available in the Cycle II data. Moreover since the precise locality of each observation cannot be identified at the present time,[29] it is not possible to estimate these prices with local market data. Therefore to partially control for these prices, we use a set of three region and four size of place of residence variables. (These variables will also control for other regional differences that are not otherwise accounted for.) In addition, information about the last time the child visited a dentist (DENT12 and DENTIST3) is used to provide a somewhat more specific index of the price and availability of medical care. The latter variables not only proxy the price and availability of medical care in the area but also the family's preferences and attitudes concerning preventive care.[30]

Finally, family size is represented by the number of people in the family who are under 20 years of age at the time of the Cycle II interview (LESS20). LESS20 may therefore overstate or understate actual completed family size.

In surveying these variables, it is immediately obvious that some of them are not the exogenous measures called for by the theory but rather are proxies that are in part endogenously determined. That is, some of these variables not only represent exogenous prices or endowments but are themselves outcomes of the family's decision making. For example, mother's labor force status represents her wage rate as well as the amount of time she chooses to spend with her children. Similarly, some of the health endowment measures--birth weight, mother's age at the time of the child's birth, and whether or not congenital abnormalities are present--reflect not only genetic endowments but also family choices regarding prenatal

care and the timing of childbearing, both of which condition the birth outcome. The same observation can be made about the measures used to proxy the price and availability of medical care (DENT12 and DENTIST3). They represent both exogenous prices and endogenous health inputs.

One other explanatory variable is endogenous--family size--but for a different reason. Family size is endogenous not because it is an imperfect proxy for a truly exogenous variable, but because in the theoretical model both family size and children's health are simultaneously determined.[31]

The endogeneity of family size as well as the other variables mentioned above poses the usual problem of bias in their coefficient estimates unless appropriate simultaneous estimation techniques are used. Such techniques cannot be used in this case, however, because appropriate data are not available, and even if they were, the coefficients are not always identifiable.[32] As an alternative procedure, we estimate the children's health equations two ways, both with and without the set of endogenous variables included. When they are excluded, the coefficients of the various exogenous family characteristics will reflect their gross impacts. Estimates with the endogenous input, endowment, and family size variables included will reveal to what extent the exogenous family characteristics operate through these endogenous measures.

To recapitulate, the equations we estimate have the health variables listed in Table II as dependent variables and the variables listed in Table III as explanatory variables. These equations are not pure reduced-form demand equations for children's health for two reasons. First, the inclusion of family size, an endogenous variable from the structural demand equation, makes the estimating equation a hybrid of a reduced form and structural demand equation. Second, measures of some of the relevant

theoretical variables are unavailable and the proxy measures used also represent endogenous input quantities. The final estimating equation reported on here, then, is a mixture of a structural demand equation, a reduced form demand equation, and a production function. Interpretation of our results will allow for the hybrid nature of these equations.

IV. Empirical Results

The equations described in the previous section are estimated using data for white children who live with either both of their parents or with their mothers only. There were 5,768 such children in the Cycle II sample. The exclusion of children for whom there were missing data brings the final sample to 4,196.[33] Data for both types of family composition are pooled for analysis because preliminary estimates indicated that there were no significant differences in the sets of slope coefficients for all dependent variables except SCHABS. A dummy variable identifying children who live with their mothers only (NOFATH) is included to allow for differences in children's health that may be uniquely associated with the absence of a father. We do not use data for black children in estimating the equations because preliminary analysis revealed significant race differences in slope coefficients for about half of our health measures. In addition, since the black sample is too small to allow for reliable coefficient estimates, separate estimates for black children are not presented.

The method of estimation is ordinary least squares. Although this is not the optimal econometric procedure to use when the dependent variable is dichotomous, use of LOGIT[35] or another appropriate nonlinear estimation procedure is not feasible given the sample size and number of variables in our empirical work. To determine whether this misspecification is likely to greatly affect our results, we experimented with alternative estimation procedures on a subsample of one-third of our basic sample. Using a dependent variables with a low incidence and one with an incidence near .5, we obtained both OLS and LOGIT estimates of our equations for this subsample. In both cases the differences between alternate estimators are small: OLS coefficients and the analogous marginal effects in LOGIT are of similar magnitudes and signs, and the patterns of statistical significance do not alter. Indeed, the differences between these OLS and LOGIT estimates for the one-third subsample are much less than are the differences between OLS estimates for the full sample and for the one-third subsample. On the basis of these experiments we believe that OLS estimation applied to the full sample provides the more accurate picture of the relationship between measures of health and the various explanatory variables. Of course, the usual statistical tests on a single coefficient or on sets of coefficients can only be interpreted as suggestive when the dependent variable is dichotomous because the assumptions underlying these tests are not satisfied.

We organize our discussion of the results around groups of explanatory variables. Therefore, rather than presenting estimates of the equations in a single massive table, we have chosen to partition the results into several tables, each showing the coefficients of a subset of explanatory variables.

It should be emphasized, however, that the coefficients in each of these tables are taken from multiple regression estimates that contain either the complete set of explanatory variables in Table III or that subset we have classified as exogenous.

A. Overview

The obvious first question to ask is are these health measures amenable to statistical explanation with the set of variables considered here? To answer this question we present adjusted R^2's and "F" statistics for the health equations both with and without the set of endogenous explanatory variables (Table IV). In all cases except for hearing acuity and excessive school absence the equations are statistically significant at the 1 percent level of significance in one or both formulations. Further, only for the hearing variable are none of the individual explanatory variables ever statistically significant. Thus, we can conclude that for most of the health measures we study, observed variations in our sample are not caused solely by chance but rather are systematically related to the set of family, endowment, and region and city size characteristics considered here.

B. Exogenous Family Characteristics

Since the prime focus of this paper is the relationship between family characteristics and children's health, we begin by looking at the measures of exogenous family characteristics--income, parents' education, whether or not a father lives with the family, and whether or not a foreign language is spoken in the home. The coefficients of these variables appear in Table V (the results for IHEAR are not shown in this and

TABLE IV
Adjusted R Squares and F Statistics

Dependent Variable	Equation 1[a] Adjusted R Squared	Equation 1[a] F Statistic	Equation 2[b] Adjusted R Squared	Equation 2[b] F Statistic
PFGHEALTH	.073	(21.71*)	.083	(15.61*)
IHEAR	-.001	(0.73)	-.002	(0.65)
ABVIS	.005	(2.26*)	.006	(1.99*)
HDBP	.006	(2.79*)	.007	(2.25*)
ALLEG	.032	(9.69*)	.037	(7.15*)
TENS	.020	(6.32*)	.020	(4.35*)
ACABN	.002	(1.62)	.006	(1.89*)
SCHABS	.001	(1.34)	.002	(1.22)
IHEIGHT	.040	(12.63*)	.071	(13.75*)
IWEIGHT	.030	(9.60*)	.060	(11.72*)
APERI	.104	(33.50*)	.109	(21.55*)

*Statistically significant at the 1 percent level of significance.

[a]Explanatory variables are FINC, MFINC, FEDUCAT, MEDUCAT, FLANG, NOFATH, MALE (when relevant), FIRST, TWIN, NEAST, MWEST, SOUTH, URB1, URB2, URB3 and NURB.

[b]Explanatory variables include those in equation 1 plus LESS20, LIGHT1, LIGHT2, CABN, MWORKPT, MWORKFT, LMAG, MMAG, DENT12 and DENTIST3.

TABLE V

Coefficients of Exogenous Family and Child Characteristics*
("F" statistics in parentheses)

Explanatory Variable	PFGHEALTH Eq. (1)	PFGHEALTH Eq. (2)	ABVIS Eq. (1)	ABVIS Eq. (2)	HDBP Eq. (1)	HDBP Eq. (2)	ALLEG Eq. (1)	ALLEG Eq. (2)	TENS Eq. (1)	TENS Eq. (2)
FINC	-.0121	-.0115	-.0028	-.0035	.0026	.0023	.0022	.0008	.0011	.0013
	(5.75)	(5.17)	(0.70)	(1.06)	(1.17)	(0.92)	(0.34)	(0.04)	(0.04)	(0.06)
HFINC	-.0005	-.00004	.0033	.0035	-.0015	-.0016	.0003	.0005	-.0021	-.0024
	(0.02)	(0.00)	(2.06)	(2.26)	(0.85)	(0.99)	(0.01)	(0.04)	(0.35)	(0.47)
FEDUCAT	-.0139	-.0118	.0038	.0037	-.0031	-.0031	.0059	.0053	-.0032	-.0035
	(19.68)	(14.14)	(3.32)	(3.12)	(4.50)	(4.49)	(6.44)	(5.14)	(0.96)	(1.14)
MEDUCAT	-.0110	-.0080	-.0036	-.0046	-.0022	-.0029	.0100	.0072	.0061	.0068
	(9.21)	(4.65)	(2.21)	(3.40)	(1.70)	(2.73)	(13.87)	(6.66)	(2.71)	(3.07)
FLANG	-.0048	-.0121	-.0127	-.0121	-.0136	-.0139	-.0179	-.0150	-.0734	-.0739
	(0.04)	(0.22)	(0.55)	(0.49)	(1.27)	(1.31)	(0.88)	(0.61)	(7.66)	(7.72)
NOFATH	-.0019	-.0179	-.0231	-.0277	-.0238	-.0253	-.0124	-.0167	-.0291	-.0208
	(0.004)	(0.33)	(1.30)	(1.78)	(2.77)	(2.97)	(0.30)	(0.52)	(0.86)	(0.42)
FIRST	-.0544	-.0342	.0110	.0054	.0090	.0086	.0426	.0247	.1240	.1266
	(10.86)	(3.51)	(1.00)	(0.20)	(1.35)	(0.99)	(12.01)	(3.28)	(52.98)	(44.68)
TWIN	.0035	-.0262	.0407	.0396	-.0218	-.0269	-.0982	-.0881	.0177	-.0062
	(0.01)	(0.26)	(1.49)	(1.33)	(0.86)	(1.24)	(6.95)	(5.32)	(0.12)	(0.01)
MALE	-.0014	-.0024	-.0250	-.0251	-	-	.0324	.0336	.0568	.0573
	(0.01)	(0.03)	(6.39)	(6.38)	-	-	(8.60)	(9.24)	(13.75)	(13.96)

(continued on next page)

TABLE V (concluded)

Explanatory Variable	ACABN		SCHABS		IHEIGHT		IWEIGHT		APERI	
	Eq. (1)	Eq. (2)	Eq. (1)	Eq. (2)	Eq. (1)	Eq. (2)	Eq. (1)	Eq. (2)	Eq. (1)	Eq. (2)
FINC	.0020 (1.03)	.0020 (0.99)	.0043 (3.52)	.0044 (3.77)	.0040 (0.16)	.0006 (0.004)	.0237 (5.27)	.0191 (3.50)	-.0175 (5.37)	-.0167 (4.89)
HFINC	-.0019 (1.97)	-.0018 (1.79)	-.0036 (5.30)	-.0036 (5.41)	.0077 (1.26)	.0069 (1.05)	-.0080 (1.29)	-.0075 (1.16)	.0045 (0.77)	.0047 (0.82)
FEDUCAT	.0016 (1.74)	.0020 (2.67)	.0011 (0.55)	.0013 (0.80)	.0155 (6.16)	.0113 (3.32)	.0073 (1.31)	.0060 (0.90)	-.0241 (26.57)	-.0209 (19.61)
MEDUCAT	-.0007 (0.24)	-.0004 (0.08)	-.0023 (1.89)	-.0017 (0.96)	.0257 (12.75)	.0134 (3.37)	.0152 (4.21)	.0033 (0.20)	-.0218 (16.24)	-.0177 (10.07)
FLANG	.0254 (6.41)	.0233 (5.37)	-.0122 (1.11)	-.0138 (1.41)	-.0666 (1.69)	-.0584 (1.33)	.0279 (0.28)	.0287 (0.31)	-.0099 (0.07)	-.0159 (0.17)
NOFATH	-.0212 (3.18)	-.0263 (4.70)	.0285 (4.26)	.0281 (3.94)	-.0352 (0.34)	-.0263 (0.19)	-.0266 (0.18)	-.0493 (0.62)	.0701 (2.38)	.0405 (0.76)
FIRST	.0050 (0.60)	.0059 (0.69)	-.0076 (1.03)	-.0026 (0.10)	.1742 (28.04)	.1410 (15.34)	.2024 (36.01)	.1440 (15.21)	.0185 (0.56)	.0162 (0.35)
TWIN	-.0143 (0.54)	-.0207 (1.07)	.0229 (1.06)	.0108 (0.22)	-.0365 (0.13)	.1313 (1.70)	-.0341 (0.11)	.1193 (1.33)	-.0308 (0.17)	-.0428 (0.31)
MALE	-.0026 (0.20)	-.0028 (0.23)	-.0065 (0.93)	-.0059 (0.77)	- -	- -	- -	- -	- -	- -

*Coefficients are statistically significant at the 5 percent level if the "F" is greater than 3.84 for a two-tailed test, or 2.69 for a one-tailed test. The explanatory variables in Equation 1 are FINC, HFINC, FEDUCAT, MEDUCAT, FLANG, NOFATH, MALE (when relevant), FIRST, TWIN, NEAST, MWEST, SOUTH, URB1, URB2, URB3, and NURB. The explanatory variables in Equation 2 include those in Equation 1 and also LESS20, LIGHT1, LIGHT2, CABN, MWORKPT, MWORKFT, LMAG, HMAG, DENT12 and DENTIST3.

subsequent tables because none of its explanatory variable coefficients are statistically significant).

The partial effects of income on the various health measures are surprisingly small and are statistically significant only for the variables PFGHEALTH, SCHABS, IWEIGHT, and APERI. As an example of the magnitudes of these coefficients, a $1,000 increase in annual family income is associated with a decrease of only .01 in the probability of parents' rating their children's health as poorer than "very good" (see eq. 1). The magnitudes tend to become even smaller when the set of endogenous variables is included (eq. 2), but the pattern of statistical significance is not altered. In addition, in some cases higher income is associated with poorer rather than better health (HDBP, ALLEG, ACABN, TENS, SCHABS), although this perverse relationship is statistically significant only for SCHABS. With respect to differences in income effects between low and high income families, a significant difference is observed only for SCHABS. In that case the negative income effect in the low income class completely disappears for the high income class (the coefficients of FINC and HFINC are opposite in sign and approximately equal in magnitude). To summarize these varied results, while income does have a significant relationship with four of the eleven health measures, on balance, the evidence leads one to conclude that, overall, income is not an important factor for explaining health variations among the children in this sample.[36]

Unlike family income, the parents' educational attainment has significant effects on most of the health measures. In fact, one or both of the parents' education measures is statistically significant except when ACABN and SCHABS are the dependent variables. This is true whether or not the set of endogenous variables is included in all equations except the weight

equation. Both for the height and weight measures the inclusion of the endogenous variables causes a substantial (about 50 percent on average) decline in the magnitude of the education coefficients. Contrary to expectations, significant inverse relationships between health and parents' education are reported for ALLEG and TENS. This may be a result of a reporting bias in that more educated parents may be more sensitive to subtle aspects of ill health in their children. Alternatively, more educated parents may be more demanding regarding their children's behavior and achievement, creating greater tension and accompanying allergies. With the exception of these two measures of the "new morbidity," however, the coefficients of the parents' educational attainment variables are consistent with the notion that higher parental education leads to greater efficiency in home health production.[37]

Children from families where a foreign language is spoken in the home do not have significantly different health levels from other children except with respect to the measures ACABN and TENS. Children from such families are more likely to exhibit an acquired abnormality, but they are less likely to be considered tense by their parents.

Differences in children's health associated with the presence or absence of a father in the household at the time of the survey are not uniform across the various health measures, and in many cases, are not statistically significant. The coefficient of the dummy variable NOFATH indicates significant positive associations with better health when HDPB and ACABN are the health measures, and significant negative associations with better health when health is measured by school absenteeism. In the cases where NOFATH is not significant, both positive and negative health relationships are again reported.[38] The somewhat unexpected conclusion to be drawn here, then, is

that having an absent father is neither a clear health advantage nor a clear health disadvantage to a child. One possible explanation is that, with family income held constant, unmarried mothers have more resources to allocate to their own consumption and to their children than do married couples.

Among the exogenous family characteristics considered in this subsection, it is not family income that appears to be of outstanding importance in explaining variations in children's health levels (though income does play a role), but rather parents' educational attainment. With the exception of the two measures relating to the "new morbidity" (ALLEG and TENS) children's health status significantly improves as their parents' educational attainment increases. The remaining two exogenous characteristics of families--speaking a foreign language in the home and having an absent father--appear to have minimal negative impacts on children's health, and in some cases, may even be positively associated with health.

C. Region and City Size

It is clear from the coefficients of the region and city size variables (in Table VI) that another characteristic of families--their location--accounts for larger differences in children's health than do those characteristics discussed above. Significant regional differences exist for the variables PFGHEALTH, ABVIS, ALLEG, ACABN, APERI, IHEIGHT, and IWEIGHT); and significant city size differences exist for PFGHEALTH, ABVIS, HDBP, ALLEG, TENS, IWEIGHT, and APERI. Regional and city size differences in children's health are not, however, uniform; there is no "best" place to live. For example, health is best in the West when measured by APERI and ACABN, but worst when measured by ALLEG, IHEIGHT, and IWEIGHT. Similarly, health is best in non-urbanized non-rural areas when measured by HDBP, but is best in medium sized cities (1 to 3 million people) when measured by PFGHEALTH and ALLEG.

TABLE VI
Coefficients of Region and City Size Variables*
("F" statistics in parentheses)

Explanatory Variable	PFGHEALTH Eq. (1)	PFGHEALTH Eq. (2)	ABVIS Eq. (1)	ABVIS Eq. (2)	HDBP Eq. (1)	HDBP Eq. (2)	ALLEG Eq. (1)	ALLEG Eq. (2)	TENS Eq. (1)	TENS Eq. (2)
NEAST	-.0733	-.0663	.0314	.0281	.0107	.0086	-.0693	-.0759	.0029	.0005
	(10.74)	(8.72)	(4.42)	(3.48)	(1.03)	(0.65)	(17.26)	(20.41)	(0.02)	(0.00)
MWEST	-.0325	-.0302	.0145	.0114	.0042	.0032	-.0607	-.0625	-.0020	-.0052
	(2.69)	(2.30)	(1.20)	(0.73)	(0.20)	(0.12)	(16.92)	(17.65)	(0.01)	(0.06)
SOUTH	.0718	.0702	-.0057	-.0089	.0156	.0141	-.0477	-.0536	.0176	.0163
	(9.19)	(8.80)	(0.13)	(0.32)	(1.95)	(1.59)	(7.31)	(9.18)	(0.52)	(0.44)
URB1	.0093	.0161	.0161	.0148	.0110	.0097	-.0007	-.0071	.0544	.0549
	(0.17)	(0.50)	(1.12)	(0.94)	(1.05)	(0.81)	(0.002)	(0.18)	(5.33)	(5.39)
URB2	-.0732	-.0695	.0145	.0142	-.0015	-.0021	-.0491	-.0527	.0854	.0869
	(8.13)	(7.39)	(0.72)	(0.68)	(0.02)	(0.03)	(6.57)	(7.60)	(10.38)	(10.72)
URB3	.0003	.0076	.0272	.0258	.0127	.0111	.0240	.0172	.0555	.0572
	(0.00)	(0.12)	(3.48)	(3.09)	(1.52)	(1.16)	(2.18)	(1.11)	(6.05)	(6.35)
NURB	-.0398	-.0423	.0075	.0073	-.0328	-.0317	.0068	.0050	.0371	.0407
	(2.92)	(3.31)	(0.23)	(0.22)	(8.96)	(8.32)	(0.16)	(0.08)	(2.39)	(2.85)

(continued on next page)

TABLE VI (concluded)

Explanatory Variable	ACABN Eq. (1)	ACABN Eq. (2)	SCHABS Eq. (1)	SCHABS Eq. (2)	IHEIGHT Eq. (1)	IHEIGHT Eq. (2)	IWEIGHT Eq. (1)	IWEIGHT Eq. (2)	APERI Eq. (1)	APERI Eq. (2)
NEAST	.0217	.0236	.0124	.0142	.0669	.0327	.0454	.0221	.4704	.4894
	(6.15)	(7.18)	(1.50)	(1.94)	(2.26)	(0.55)	(0.99)	(0.24)	(198.16)	(211.47)
MWEST	.0242	.0251	.0060	.0076	.1121	.0969	.1423	.1406	.1114	.1225
	(9.77)	(10.30)	(0.45)	(0.71)	(8.06)	(6.08)	(12.36)	(12.19)	(14.16)	(16.86)
SOUTH	.0165	.0161	-.0057	-.0050	.0100	-.0058	.0272	.0065	.3566	.3642
	(3.17)	(3.00)	(0.28)	(0.21)	(0.05)	(0.02)	(0.32)	(0.02)	(101.50)	(105.51)
URB1	-.0146	-.0135	-.0086	-.0079	.0262	.0058	.1155	.0951	-.0255	-.0175
	(2.70)	(2.28)	(0.69)	(0.58)	(0.33)	(0.02)	(6.13)	(4.26)	(0.56)	(0.26)
URB2	-.0042	-.0037	-.0017	-.0011	.0543	.0470	.1065	.0986	.0066	.0110
	(0.17)	(0.14)	(0.02)	(0.01)	(1.12)	(0.87)	(4.12)	(3.63)	(0.03)	(0.08)
URB3	-.0066	-.0053	-.0080	-.0072	.0024	-.0153	-.0828	-.0987	.0344	.0412
	(0.59)	(0.38)	(0.66)	(0.53)	(0.003)	(0.13)	(3.44)	(4.98)	(1.11)	(1.58)
NURB	.0002	-.0005	.0082	.0086	-.0499	-.0388	-.0073	-.0068	-.0387	-.0436
	(0.001)	(0.003)	(0.61)	(0.68)	(1.16)	(0.72)	(0.02)	(0.02)	(1.24)	(1.57)

*See Table V.

The reasons for these differences are varied and cannot be explored with the types of data used in this paper. We suggest a number of possibilities: climate, air pollution levels, fluoridation levels, ethnic composition of the population, as well as unmeasured differences in the availability and price of medical care. In addition, there are differences in stress and tension associated with living in cities of various sizes or in rural areas. To distinguish between these explanations, one would need to match up the data in the Cycle II survey with local measures of variables like those suggested above. The point we wish to emphasize here, however, is that even when a large number of individual family demographic and economic characteristics are held constant, large unexplained regional and city size differences in children's health levels exist. These unexplained differences are clearly an important issue for future epidemiological research.

D. Exogenous Child Endowments

Coefficient estimates for the three exogenous child endowment characteristics--FIRST, TWIN, and MALE--appear at the bottom of Table V. First born children have significantly better health when measured by PFGHEALTH, IHEIGHT, and IWEIGHT, and significantly poorer health when measured by ALLEG and TENS. Additional insight regarding the role of this endowment measure is obtained by looking at how its coefficient alters when the set of endogenous variables is included. In this case both the benefits and disadvantages of being a first born child diminish substantially (except for TENS), though FIRST remains statistically significant. Thus, the impact on child health of being a first born operates in part via such endogenous variables as family size, mother's work status, and dentist visits.

These reported health differences between first borns and others are consistent with the notion that first born children receive relatively more time and attention from their parents than other children: their rate of physical development is more rapid (despite the fact that they tend to have a lower weight at birth), but at the same time they exhibit a greater incidence of allergies and tension problems. The latter is a likely result of the increased parental attention (and accompanying expectations) directed at first borns.

With respect to the other two child characteristics, male children are less likely to have poor vision and more likely to have allergies and be tense than are female children. These sex differences remain unaffected by the inclusion of the endogenous variables. Twins differ significantly from other children in their health status only in that they are less likely to have allergies. To conclude, there are health differences associated with these exogenous child characteristics, in particular with being a first born child, although these differences are not uniform across the various measures of health.

E. Endogenous Child Health Endowments

The coefficients of the five endogenous measures of the child's health endowment appear in Table VII. Three of these measures reflect differences in the birth outcome (LIGHT1, LIGHT2, CABN), and the remaining two indicate the mother's age at the time of the child's birth (LMAG, HMAG). One or more of the three birth outcome variables is significantly associated with poorer current health for the measures PFGHEALTH, ABVIS, ACABN, SCHABS, IHEIGHT, IWEIGHT, and APERI. Especially notable are the effects of these variables on height and weight: babies of low birth weight have subsequent height

TABLE VII
Coefficients of Family Size and of Endogenous Endowments*
("F" statistics in parentheses)

Explanatory Variable	PFGHEALTH	ABVIS	HDBP	ALLEG	TENS	ACABN	SCHABS	IHEIGHT	IWEIGHT	APERI
LIGHT1	.0043 (0.004)	.0367 (0.66)	.0448 (1.99)	.0006 (0.000)	.1065 (2.33)	.0584 (4.91)	.0600 (3.81)	-.5135 (15.02)	-.3918 (8.32)	.0928 (0.85)
LIGHT2	.1113 (9.08)	-.0102 (0.17)	-.0001 (0.00)	.0003 (0.000)	.0489 (1.63)	-.0005 (0.001)	.0156 (0.86)	-.4529 (38.63)	-.3830 (26.27)	-.0203 (0.13)
CABN	.0534 (2.48)	.0590 (6.73)	.0057 (0.13)	.0068 (0.07)	.0504 (2.05)	.0381 (8.21)	-.0172 (1.20)	-.1506 (5.08)	-.1551 (5.13)	.0896 (3.12)
LMAG	-.0557 (3.42)	-.0034 (0.03)	-.0113 (0.63)	-.0024 (0.01)	-.0156 (0.25)	-.0089 (0.57)	-.0125 (0.79)	-.1783 (9.01)	-.1860 (9.32)	.1229 (7.41)
HMAG	.0992 (4.72)	.0132 (0.65)	.0222 (3.68)	-.0100 (0.30)	.0305 (1.44)	-.0004 (0.002)	.0090 (0.66)	.1315 (7.43)	.1716 (12.04)	.0169 (0.21)
LESS20	.0067 (1.85)	-.0049 (2.20)	-.0024 (1.11)	-.0139 (14.32)	-.0009 (0.03)	-.0009 (0.22)	.0020 (0.80)	-.0536 (30.70)	-.0079 (61.51)	.0110 (2.24)

*Coefficients are statistically significant at the 5 percent level if the "F" is greater than 3.84 for a two-tailed test, or 2.69 for a one-tailed test. The explanatory variables included in the equations are as for Equation 2 (see note to Table V).

and weight of almost half a standard deviation below the mean for their age-sex cohort.

The results for the mother's age variables are less uniform. Children of young mothers are significantly healthier when health is measured by PFGHEALTH and significantly less healthy for height, weight, and the peridontal index. Conversely, children of older mothers are significantly less healthy according to the results for PFGHEALTH and HDBP and more healthy for height and weight. These observed relationships between mother's age and children's health reflect two forces that go in opposite directions. Relatively young mothers are probably in better physical health when they give birth, but they are more likely to have unwanted births and consequently receive poorer prenatal care and spend less time with their children. Births at relatively old ages pose health risks both to mothers and children, but older mothers might be more efficient in caring for their children.

As mentioned earlier, all five of these variables are endogenous in that they are determined simultaneously with the health measures. Therefore the reported significant relationship between a poor birth outcome and poorer current health can be interpreted not only as the result of behavioral or physiological relationships, but also as the impact of unmeasured factors that affect both early and current health (tastes, attitudes towards medical care, etc.). A similar remark can be made regarding the reported coefficients of the two mother's age variables.

F. Family Size

Family size has significant health effects only for height, weight, and the incidence of allergies (see Table VI). Children from larger families tend to be shorter and thinner than other children, but they also exhibit a

smaller probability of having allergies. Whether these relationships reflect quality-quantity substitution on the part of the parents or some alternative mechanism (such as the effects of an exogenous constraint on the amount of parents' time available to each child) cannot be determined.

G. Mother's Work Status

The two mother's work status variables (MWORKFT, MWORKPT) are not significantly related to most of the health measures (see Table VIII).[39] When an "F" test on the pair of variable coefficients is conducted, they have significant effects only for IHEIGHT and APERI (we do not show these tests). In both cases, children of working mothers have poorer health than do other children. The coefficients of the individual work status variables indicate that working full-time has a greater impact on the periodontal index, while working part-time has a greater impact on height. In general these results reveal that participation by mothers in the labor market is detrimental to the health of their children only with respect to two of the measures which reflect the child's nutritional status. This finding is especially plausible in that working mothers are clearly less able to supervise their children's diets.

H. Medical Care

The regression coefficients of the dichotomous variables that identify children who last saw a dentist more than one year ago (DENT12) and children who have never seen a dentist (DENTIST3) appear in Table VIII. These two variables serve as negative proxies of the amount of preventive medical care and positive proxies of the price of preventive care: parents of children who have seen a dentist within the past year are more likely to obtain preventive medical care services for their children and to face a lower price

TABLE VIII
Coefficients of Mother's Work Status and Medical Care Variables*
("F" statistics in parentheses)

Explanatory Variable	PFGHEALTH	ABVIS	HDBP	ALLEG	TENS	ACABN	SCHABS	IHEIGHT	IWEIGHT	APERI
MWORKPT	.0333 (2.24)	-.0139 (0.87)	-.0006 (0.003)	-.0023 (0.02)	-.0114 (0.25)	-.0057 (0.42)	.0001 (0.000)	-.1272 (8.41)	-.0179 (0.16)	.0075 (0.05)
MWORKFT	.0345 (2.62)	.0066 (0.21)	.0036 (0.13)	.0102 (0.41)	-.0322 (2.12)	.0123 (2.16)	.0025 (0.07)	-.0624 (2.20)	.0164 (0.15)	.0849 (7.05)
DENT12	.0992 (22.41)	-.0009 (0.004)	-.0211 (4.55)	-.0423 (7.30)	-.0263 (1.46)	.0161 (3.83)	.0026 (0.07)	-.1051 (6.47)	-.0164 (0.15)	.0726 (5.34)
DENTIST3	.0887 (15.76)	-.0227 (2.29)	-.0110 (1.08)	-.0453 (7.34)	.0012 (0.003)	.0153 (3.06)	.0156 (2.30)	-.2098 (22.65)	-.1067 (5.57)	.0897 (7.18)

*See Table VII.

of care. These parents may also have preferences for high quality children. When the periodontal index is the health measure, these two variables also proxy direct inputs in the production of dental health.

One or both of these medical care proxies are significantly related to seven of the ten health measures (all but ABVIS, TENS, and SCHABS). For five of the seven (PFGHEALTH, ACABN, IHEIGHT, IWEIGHT, and APERI) children who saw a dentist within the past year have higher levels of health than children who saw a dentist more than one year ago or children who never saw a dentist. This relationship is reversed for ALLEG and HDBP. The perverse allergy effects probably indicate a greater awareness of allergy problems among parents who took their children to a dentist within the past year. The blood pressure effect is puzzling, and we offer no explanation of it.

These preventive medical care proxies appear to have the largest impacts on health measures that reflect basic nutritional status. In the height, weight, and peridontal index equation, the coefficient of the variable that identifies children who never saw a dentist is large relative to the coefficients of other independent variables. In the height regression the coefficient of DENTIST3 equals 21 percent of the standard deviation in height. The corresponding figures in the weight and peridontal index regressions are 11 percent and 12 percent, respectively. Not only are these coefficients large, but they apply to a substantial proportion of our sample: 18 percent of the children in the sample never saw a dentist.

The statistically significant effects of the two dental variables on the periodontal index are particularly noteworthy since in this case our estimates directly measure the effect of oral health input on oral health output. In fact, for this health measure the beneficial input effects

that we report are likely to understate true input effects because of reverse causality that runs from a reduction in oral health to an increase in the probability of contacting a dentist.

V. Income Differences in Children's Health

Much of the attention policy makers and health professionals direct at children's health focuses on differences related to family income levels. In this section, we discuss how our results can be used to provide additional insight into the nature and causes of these differences.

Our findings of small, and, in most cases, nonsignificant income effects on children's health may appear to be at variance with accepted opinion. Indeed, in our introduction we refer to large reported differences in health associated with differences in family income. One explanation of these contradicting conclusions is that the "conventional wisdom" is based to a large extent on income differentials in infant mortality and low birth weight. Our paper does not deal with infant mortality, and low birth weight is treated as an explanatory variable rather than one to be explained. Moreover, our sample consists of a cohort of children who survived beyond the first year of life so that a substantial proportion of low birth weight infants are not included. [It is well known that low birth weight has a strong positive relationship with subsequent infant mortality [see Lewit 1977].

In addressing the health of children in mid-childhood, we find that while for some health measures simple income comparisons do reveal large differences between the health of children from low and high income families, these differences largely disappear when one controls for differences in

other family characteristics that are highly correlated with income. To demonstrate how large a portion of the apparent (or gross) income differences would be attributed to associated family characteristics, in Table IX we provide illustrative calculations for the measures PFGHEALTH, IHEIGHT, IWEIGHT, ALLEG, and APERI. These are the only health measures for which statistically significant gross income differences are observed in our sample. The third column of Table IX shows the gross differences in these measures between children in families with annual income under $5,000 and those in families with annual income of $5,000 or more. A $5,000 family income cutoff is selected because it identifies the lowest quartile of the income distribution. The gross differences in column (3) are to be compared to health differences that are allocated to income when all other exogenous explanatory variables in our basic equations are held constant [column (4)]. The "net" income effects are less than one-half (and in some cases, one-quarter) of the observed gross income differences for these five health measures. The difference between net and gross income effects becomes even larger when the set of endogenous variables is also held constant [column (5)]. The conclusion to be drawn is clear: gross income differences in health greatly overstate the true relationship between family income and health.

If the reported gross income differences are not primarily a result of differences in income, what does account for them? To answer this question we calculate how much of the gross high-low income differences in the above five health variables can be attributed to specific explanatory variables or sets of these variables. The procedure is simply to multiply the coefficients of these explanatory variables by the differences in their mean values in the high and low family income samples of children. The resulting

TABLE IX

Income Differences in Selected Current Health Measures

Variable	Mean, Family Income ≥ $5,000	Mean, Family Income < $5,000	Gross Difference[a]	Net Difference[b]	
				Equation (1)	Equation (2)
PFGHEALTH	.394	.589	-.195	-.079	-.072
IHEIGHT	.099	-.181	.280	.079	.053
IWEIGHT	.106	-.114	.220	.091	.066
APERI	-.097	.117	-.214	-.077	-.071
ALLEG	.176	.108	.068	.016	.008

[a]Gross difference equals column (1) minus column (2). As shown in Table X, the gross differences vary little from differences that are predicted on the basis of a regression of the health variables on all the independent variables.

[b]Net difference is the difference in mean health levels between the two income classes predicted on the basis of the income coefficients in Table V. LESS20, LIGHT1, LIGHT2, CABN, MWORKPT, MWORKFT, LMAG, HMAG, DENT12 and DENTIST3 are excluded for equation (1) and included for equation (2).

estimates (in Table X) illustrate how much of the gross difference would disappear if the low income class is given the same mean values of the independent variables as the high income class and if the relationship between health and the explanatory variables is the same in both income classes. They also identify which explanatory variables are responsible for the sizable gap between the gross and the net income effects in Table IX.

Several results in Table X are noteworthy. First, almost all of the observed differences in the five health measures between the high and low income subsamples can be accounted for by differences in the independent variables that we have included in our empirical work [either equation (1) or (2)]. Second, a detailed examination of the decomposition that uses only the exogenous variables [equation (1)] indicates that differences in parents' schooling account for a large portion of observed gross income differences in health. Indeed, for three of the five variables--APERI, IHEIGHT, and IWEIGHT--differences in parents' average schooling between high and low income families account for a larger portion of the health differences than do differences in income. Parents' schooling remains as an important explanatory factor even when the set of endogenous variables is entered in the equation. In the latter case, however, dental care and, to a lesser extent, family size also make substantial contributions to the gross income differences in health.

TABLE X

Components of the Difference in PFGHEALTH, APERI, IHEIGHT, IWEIGHT and ALLEG Between Children from High and Low Income Families*

Component	PFGHEALTH Eq. (1)	PFGHEALTH Eq. (2)	APERI Eq. (1)	APERI Eq. (2)	IHEIGHT Eq. (1)	IHEIGHT Eq. (2)	IWEIGHT Eq. (1)	IWEIGHT Eq. (2)	ALLEG Eq. (1)	ALLEG Eq. (2)
Family income	-.079	-.072	-.077	-.071	.079	.053	.091	.066	.016	.008
Parents schooling	-.071	-.057	-.131	-.111	.115	.070	.062	.027	.045	.035
Other exogenous family characteristics	.001	.004	-.011	-.005	.012	.010	.002	.006	.004	.004
Exogenous child endowments	-.003	-.002	.001	.001	.008	.007	.010	.007	.003	.002
Region	-.025	-.024	.019	.021	.013	.010	.008	.008	-.005	-.005
City size	-.005	-.008	-.001	.001	.008	.005	.021	.017	-.004	-.005
Family size	-	-.004	-	-.006	-	.031	-	.044	-	.008
Endogenous endowments	-	.000	-	-.008	-	.014	-	.013	-	.000
Mother's work status	-	.001	-	.001	-	-.005	-	-.001	-	-.000
Dental care	-	-.031	-	-.028	-	.059	-	.026	-	.015
Total = Predicted Gross Difference	-.182	-.193	-.200	-.205	.235	.254	.194	.213	.059	.062
Actual Gross Difference		-.195		-.214		.280		.220		.068

* Only exogenous variables are in Equation 1. All variables are in Equation 2.

VI. Summary and Implications

In this study multivariate techniques have been employed to examine the determinants of eleven components of health in a national sample of white children between the ages of six and eleven. The most important empirical results and their policy implications are highlighted below.

The partial effects of family income on health are small and seldom statistically significant. Indeed, some health problems--high blood pressure, allergies, and tension--are more likely to occur among children from high income families. This phenomenon can be viewed as the early forerunner of the positive relationship between income and morbidity and mortality rates observed for adults in the United States [for example, Auster, et al. 1969 and Grossman 1972 and 1975]. The general finding of small partial income effects is supported by analysis of gross health differences between children from lower (under $5,000 per annum) and higher ($5,000 per annum and over) income families. In those cases where significant gross health differences do exist between children from these two income classes, decomposition of these gross differences shows them to be attributable in large part to exogenous factors other than income itself.

In contrast to family income, parents' schooling is an important determinant of children's health. In most instances children of well educated parents are in better health than those of less well educated parents. In fact, for four of the five health measures that have a significant gross correlation with income, much of this observed income difference is accounted for by associated differences in parents' schooling. This would suggest that policies to raise parents' schooling would not only benefit their children's health, but would also reduce differences in health between children from low and high income families.

The other exogenous family characteristics studied--the absence of a father in the home and the use of a foreign language in the home--have at best small impacts on children's health. A similar conclusion is to be drawn regarding the child's exogenous endowments--his sex, twin status, and whether he is a first-born--with the exception of the latter. First born children do have some health advantages, but they also rate more poorly than other children when health factors related to the new morbidity (allergies and tension) are examined.

The final type of exogenous variables studied describe the region and urban-rural characteristics of the child's residence. Our major finding with respect to these variables is that locational factors play an important, but largely unexplained, role in determining children's health levels. This finding emphasizes the need for additional research investigating the source of these striking locational effects on children's health.

Among the various endogenous variables studied, our most interesting results are for the variables representing mother's labor force status, family size, and the frequency with which the child received dental care. The mother's labor force status and family size variables are interesting primarily because of their lack of importance. That is, both of these factors have small health effects and are strongly related only to the health variables representing the child's nutritional status (height, weight, and the periodontal index). Children whose mothers are in the labor force or who come from larger families are likely to score more poorly with respect to these nutritional measures. In contrast with the roles of mother's labor force status and family size, the dental care variables have large and significant impacts for most of the health measures. Interpretation of this finding is not altogether clear-cut, however, since the dental care

variables proxy not only the price and availability of medical care, but also family attitudes towards preventive health care and towards health in general.

The implications of our findings are at the same time both heartening and disheartening. For example, the finding that differences in health related solely to income are smaller than commonly believed implies that policies that aim to improve the well-being of children via income transfers, such as those advocated by the recent Carnegie Council on Children [Keniston 1977] would have, at best, very small effects on health. A related implication pertains to proposals by Newberger, et al. 1976, Keniston 1977, and Marmor 1977 to restrict national health insurance to rather complete prenatal and pediatric care coverage to offset variations in health associated with income. Again, our results indicate that there is not much to offset, even though pediatric care utilization is very sensitive to family income [Colle and Grossman 1978]. At the same time, however, our findings regarding the important role of the dental care variables suggest that policies directed at either improving the availability of medical care or altering public attitudes towards preventive care could have large health payoffs for children in all types of families.

Other favorable implications of our findings relate to three recent striking trends in the demography of U.S. families. These trends are the increase in the proportion of families headed by women, the increase in the labor force participation rate of married women with children, and the

reduction in family size. We find that the absence of a father in the household has little impact on children's health. Therefore, while the recent rise in the divorce rate might affect certain dimensions of children's well-being, health does not appear to be one of them. The same comment applies to the increase in labor force participation rates of married women with the exception that with respect to our two best measures of long-run nutritional status--height and the periodontal index--children rate more poorly if the mother works. Since, however, height is negatively related to family size, the detrimental impacts of increases in labor force participation rates are offset to some extent by the beneficial impacts of reductions in family size.

The implications of our results regarding parents' schooling may appear to be clear-cut at face value since parents' schooling plays such an important role in determining children's health. Caution and more research are required, however, before actually applying them to schooling policies. First, the differences in years of parents' schooling between the high and low income samples are very large (three years both for mothers and fathers), and these would probably be extremely costly to eliminate. But more important, the mechanisms by which parents' schooling affects children's health still are not well known. Consider, for example, the finding that parents' schooling is an important determinant of children's height. This result has a very definite policy implication if the mechanism at work is a positive correlation between schooling and nutritional intakes or between schooling and the knowledge of what constitutes an appropriate diet. The policy implication is much less clear-cut if the mechanism at work is a positive relationship between parents' schooling and genetic inheritance that is not fully captured by the exogenous endowment and early health variables in the

regressions. Clearly, more research on the exact role of parents' schooling is needed.

The diverse findings in this study underscore the multidimensional nature of children's health. In fact, our results illustrate how the use of a single index could be misleading since the various family characteristics can have positive impacts on some components of good health and negative impacts on others. For example, an important distinction is found between the relationship of parents income and education with the more traditional health measures (height, the periodontal index) as compared to measures of the "new morbidity" (the presence of allergies and tension). These two family characteristics have positive impacts on the traditional measures but negative impacts on measures of the new morbidity. One can speculate that the likely upward trend in the new morbidity will lead to more utilization of physicians' services for problems that are in many cases not amenable to treatment by physicians, suggesting that some modification in the training received by pediatricians and in the delivery of pediatric care services would be desirable.

Finally, although we do not altogether resist speculating about the implications of our empirical findings, we fully understand that this study falls squarely within the sphere of traditional epidemiological research. We document the statistical relationships between family characteristics and measures of children's health in detail, but even though we outline the possible role of economic factors, we cannot determine the exact nature of the mechanisms that generate these relationships. Nor was it possible to establish the causal nature of these relationships in a definitive sense. One important and unambiguous conclusion of our study, however, is that the present tendency to base government child health

programs on simplistic notions that income is the primary source of differences in children's health will not lead towards fruitful or successful public policy regarding children's health.

FOOTNOTES

[1]This change in emphasis is partially the result of the Coleman Report and subsequent research, which showed that the effect of school quality on children's achievement may be small relative to the effects of family characteristics. Averch, et al. (1972) provide an excellent review of this literature.

[2]Most states require children to receive certain immunizations before they may begin attending school. An exception to the text statement is Connecticut, which requires school children to have physical examinations every three years [Foltz and Brown 1975].

[3]This definition of the new morbidity is quoted from Haggerty, et al. 1975, p. 316.

[4]This point is made by Starfield (1977) who emphasizes that although many persons have studied the effects of medical care and socioeconomic characteristics on infant mortality, relatively few have examined the effects of these variables on the health of children who survive the first year of life. For a few recent exceptions, see Kaplan, et al. (1972); Hu (1973); Kessner (1974); Haggerty, et al. (1975); and Inman (1976).

[5]This evidence comes from Cycles II and III of the Health Examination Survey and is reported in NCHS (1973), pp. 22-26.

[6] A full description of the sample, the sampling technique, and the data collection is presented in NCHS (1967). The one deficiency of this sample from the point of view of studying children's health is the exclusion of children in institutions. To the extent that these children are more likely to have serious and disabling physical conditions, the reported incidence of certain conditions will be lower in our sample than in the entire population of children. In addition, if the probability of the institutionalization of a child with a given condition depends on the various family characteristics studied here, our results will incorporate unknown biases. The number of institutionalized children is small, however, at about four tenths of a percent of all children aged 5 through 13 years. [This is the proportion of 5-13 year-olds living in "group quarters" in 1970 according to the U.S. Bureau of the Census (1973), Tables 52 and 205. The corresponding percentages by race are .38 percent of whites and .7 percent for blacks.]

[7] Introducing uncertainty about the number and quality of children complicates the model, but many of the basic insights provided by the notion of both a quantity and quality dimension of children remain valid. Ben-Porath and Welch (1976) illustrate how uncertainty regarding one aspect of quality--the child's sex--affects fertility

[8] Recent medical advances allow some types of poor genetic endowments to be detected inter utero and defective fetuses aborted, so that parents can now partially control their children's genetic endowments.

[9]See Willis (1973) and Becker and Lewis (1973) for a full development of these points.

[10]Associated with this output demand function are input demand functions for parents' time, medical care, and other market health inputs. These input demand functions will not be studied here.

[11]Presumably, parents' education raises efficiency in the production of many household commodities. Therefore, there is an "own price effect" due to an improvement in the efficiency of producing children's health and a "cross price effect" due to an improvement in the efficiency of producing other commodities. The statement in the text with respect to the impact of parents' education on children's health assumes that the "own price effect" outweighs the "cross price effect" if the two effects go in opposite directions.

[12]The model outlined above is not directed at explaining variations in a child's health during his childhood, but rather treats child health as a single datum--his permanent health measured, say, at the end of his childhood or as an average over his childhood. If one wanted to investigate changes in health during childhood, one would want to develop a model which explicitly examines how patterns of health and health investments over childhood are determined by life cycle variations in both the prices of health inputs and the marginal products of health investments.

[13]Edwards and Grossman (1977 and 1979) use this model to study children's health and intellectual development, Leibowitz and Friedman (1979) use it to study health inputs, Tomes (1978) uses it to examine years of schooling attained. and Ishikawa (1975) uses it to explain intergenerational transfers of education

and financial wealth. Both Ishikawa (1975) and Leibowitz and Friedman (1979) treat family size as exogenous.

[14]See, for example Sullivan (1966), Berg (1973), and, more recently, Ware (1976).

[15]See Grossman (1972), p. 58. This definition is also very similar to that proposed in Torrance (1976).

[16]A good discussion of the subsidiary issue of how one measures disability in children can be found in Schack and Starfield (1973).

[17]Of course, there is a positive relationship between the two in the sense that a child with low health capital is more likely to contract some acute conditions and to have them for a more extended time period. For example, Birch and Gussow (1970) discuss how nutrition (which is clearly a determinant of "permanent" health status) and disease are intimately related.

[18]The earlier theoretical discussion pertains to children's permanent health status measured at the end of their childhood, while health measures in the Cycle II data are for 6 to 11 year-old children. Our analysis implicitly assumes that health in mid-childhood is a good proxy for the health stocks at the end of childhood.

[19]In earlier work some attempts were made to condense the health information using principal component analysis. The analysis yielded almost as many equally weighted components as there were initial health measures.

[20]The studies we consulted are: Wallace (1962); Mechanic (1964); Mindlin and Lobach (1971); Talbot, et al. (1971); Kaplan, et al. (1972); Hu (1973); Schack and Starfield (1973); Kessner (1974); Haggerty, et al. (1975); and Inman (1976).

[21]The following physicians gave us extremely helpful advice. John McNamara, M.D., then Assistant Professor of Public Health and Pediatrics at Columbia University School of Public Health and Associate Commissioner in the New York City Department of Health; Roy Brown, M.D., Associate Professor of Community Medicine and Pediatrics at the Mount Sinai School of Medicine of the City University of New York; Thomas Travers, D.D.S., Director of Ambulatory Care in the New York City Department of Health; and Ruth T. Gross, Professor of Pediatrics and Director of Ambulatory Pediatrics, Stanford University Medical Center, Stanford, California.

[22]If the actual height or weight of each age-sex group is normally distributed, IHEIGHT and IWEIGHT could be translated directly into the child's height or weight percentile. In addition to the continuous height and weight measures, we also experimented with discrete measures identifying children who are more than two standard deviations from the mean height or weight for their age-sex cohort. These measures were used to allow for non-continuous relationships between height (or weight) and family characteristics. For example, beyond some weight level, problems with obesity start to develop, so that more weight is no longer better than less weight. Results using these discrete measures did not differ greatly from those based on using IHEIGHT and IWEIGHT, so we do not report them here.

[23]One might argue that corrected rather than uncorrected vision is the appropriate measure to use here. Unfortunately, information about corrected

vision is not available in the Cycle II data. Information about whether or not the child wears glasses is available, but it is not clear that the glasses he wears actually correct his vision defect. Kessner (1974), for example, finds that 40 percent of children in a low income sample who were tested with their glasses failed a visual acuity test.

[24]In defining ACABN, we exclude abnormalities resulting from accidents or injuries because these are likely to reflect transitory rather than permanent health variations.

[25]The periodontal index suffers from the defect that it is subject to intra-rater and inter-rater variability. We have experimented with a somewhat more objective measure of oral health, the number of decayed permanent and primary teeth adjusted for age and sex, and have obtained results similar to those for the peridontal index. Compared to the number of decayed teeth, the periodontal index reflects more serious oral health problems.

[26]There is no school form for approximately 500 children in the Cycle II data set. Since excessive absence due to illness is the only variable taken from the school form, children without the school form are eliminated from the empirical analysis only when school absence is the dependent variable.

[27]A $7,000 cutoff point is used because it is most consistent with available evidence on the distribution of inheritances across families. See Edwards and Grossman (1977) for an elaboration of this point. Note that the family income measure is an imperfect measure of long-run income and has an endogenous component because it does not hold constant the father's experience and the mother's labor force status. Therefore, we experimented with an income measure that held these two factors constant. The adjusted income

variable was very highly correlated with FINC (the correlation coefficient was greater than .99) and the regression results were not altered when adjusted income was used in place of FINC. Consequently, we report results based on the use of FINC in this paper.

[28]See, for example, Birch and Gussow (1970). Cycle II does not distinguish children who are born prematurely, so we cannot determine to what extent low birth weight is a result of prematurity or of other factors.

[29]In the future the National Center for Health Statistics might provide us with area-specific input availability and price measures. This will enable us to examine the effects of medical care prices on health outputs.

[30]Information about the time of the child's last visit to a doctor is also available in the data, but would be greatly contaminated by the child's health level. We refer to the well-known reverse causality between health and medical care.

[31]In actuality even some of the exogenous variables may not be truly exogenous. For example, women who plan to have large families will be less likely to make large investments in their own education. Or, families for whom children's health is an important component of child quality may choose to live in healthier (non-urban) areas of the country. Similarly, men who have a high preference for children may choose less intensive jobs--which presumably yield lower wages--so that they can spend more time with their children.

[32]In the case of the family size variable, for example, it is very difficult to identify exogenous variables that enter the family size

structural equation or the children's health structural equation but not both.

[33]Missing information for birth weight (818 observations), FLANG (324 observations), and income (290 observations) account for most of the missing observations. In addition, children who turned twelve years old between the time the sample was chosen and the time of the interview were also excluded. (There were 72 such children in the entire Cycle II sample.)

[34]Significant race differences were found for the variables IHEIGHT, IWEIGHT, PPGHEALTH, ALLEG, and APERI.

[35]See Nerlove and Press (1973) for a description of this technique.

[36]For the four health measures for which we find significant income effects, we also experimented with a specification for the income variable which allows for five discrete income effects rather than two continuous income effects. In particular, the variables FINC and HFINC were replaced with four dummy variables which distinguish between five income classes: less than $5,000; $5,000 to $6,999; $7,000 to $9,999; $10,000 to $14,999; and $15,000 and above. We do not show the results of this specification because it yields only one additional insight: for the highest income class income never has a significant marginal impact on health, while it does for one or more of the lower income classes. The coefficient estimates for the other explanatory variables are not sensitive to changes in the specification of the income variable.

[37]One might predict that mother's education would be more important in explaining variations in children's health than is father's education since most child care is done by the mother. We do not consistently observe this

in our results. One reason might be the high correlation between mother's and father's education (r = .67 in mother-father families).

[38]These estimates "maximize" the effect of not having a father in the sense that they are derived under the extreme assumption that father's education has the same relationship with child health whether or not the father is actually present (and that the average educational attainment of absent fathers is the same as for fathers living with their families). These assumptions are needed to obtain estimates of the relationship between NOFATH and the various health measures because there is no information in our data on the educational attainment of fathers who do not currently live with their children. Use of the alternative extreme assumption, that there is no relationship between father's education and child health for children whose fathers are absent from the home, generates "minimum" estimates of the effect of having an absent father. These "minimum" estimates also indicate that NOFATH has both positive and negative relationships with better health, depending on the health measure used: significant positive health relationships with NOFATH are reported for PPGHEALTH, HDBP, and APERI; and a significant negative relationship is reported for SCHABS.

[39]This lack of significance is especially notable because the endogenous nature of the mother's work status variables biases their coefficients towards having a larger negative relationship with child health than they would have simply as measures of the mother's opportunity cost of time.

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Chapter 3

INCOME AND RACE DIFFERENCES IN CHILDREN'S HEALTH

I. Introduction

Recent studies of children's health in the United States have stressed the differences between black and white children and between children in high and low income families. Newberger, Newberger, and Richmond (1976), for example, cited mortality rates for both infants and older children that are over 50 percent higher for blacks than for whites (pp. 252-53). They also point out that the incidence of low birth weight (a good negative indicator of whether an infant will survive his first year and of his successful future development) is more prevalent among black and poor families. Similar statistics are cited by Keniston (1977) and the National Research Council (1976).

The income and race comparisons in children's health status cited above, as well as those cited elsewhere, rely primarily on measures that relate to the first year of the child's life. This results largely from the unavailability of comprehensive measures of morbidity for older children. Recent publication of data from Cycles II and III of the Health Examination Survey, however, makes it possible to explicitly study the health of older children. In this study we use data from Cycle II, which covers children aged 6 through 11 years, to explore income and race differences in nine measures of children's health.

We report two kinds of results. First, we show that when a variety of health measures from mid-childhood are the subject of analysis, both income and race differences are much less pronounced than they are in infant mortality data. Second, these differences do not uniformly favor

children from white or higher-income families. Indeed, children from poor families and black families are found to be in better rather than worse health when measures that reflect the "new morbidity" are examined. This is the phrase originated by Haggerty, Roghmann, and Pless (1975) to describe "learning difficulties and school problems, behavioral disturbances, allergies, speech difficulties, visual problems, and the problems of adolescents in coping and adjusting" (p. 316). Haggerty and his colleagues observe that over this century the traditional childhood diseases have been declining in importance while health problems associated with the new morbidity have become more widespread. That is, the rising level of family income during this century has been accompanied by an increased prevalence of the new morbidity. This observation is remarkably parallel to our own findings of a positive association between family income and the incidence of certain health problems.

II. Measuring Health in the Cycle II Sample

Cycle II of the Health Examination Survey is an exceptional source of information about a national sample of 7,119 noninstitutionalized children aged 6 to 11 years in the 1963-65 period.[1] The data comprise complete medical and developmental histories of each child provided by the parent, information on family socioeconomic characteristics, birth certificate information, and a school report with data on school performance and classroom behavior provided by teachers or other school officials. Most important, there are objective measures of health from physical examinations administered by the Public Health Service. Together, the data provide an unusually detailed picture of the health of children in this cohort.

Given the unusual detail and diversity of the health data in the Cycle II survey, the choice of health status measures is not dictated by data availability but rather is an issue that must be faced directly. The problem of defining and measuring children's (and adults') health is very much an unresolved one, even among professionals in the area of public health.[2] The economist's approach is to defined health as a form of human capital which determines the amount of time available for consumption and for work in the home and labor market.[3] With this type of definition, an appropriate measure of health status over some time period would be the proportion of potential time that is actually available for the usual consumption, maintenance, and work activities. Similarly, the complementary measure of ill health would be the proportion of time lost as a result of imperfect functioning. Such disability may be relatively easy to measure when dealing with adults who are members of the labor force (a good approximation is days lost from work because of illness), but it is not easy to measure for other adults or for children. Therefore, in economists' studies of adults' health, both the incidence of particular physical conditions and the individual's own assessment of his health status have been used as supplementary health measures [Grossman (1975)].

We use the same type of restricted, morbidity-oriented, definition of children's health, focusing on the child's physical health rather than his overall well-being and on similar types of measures: disability, physical conditions, and parental assessment of health status. Even some of these measures, however, may not always be appropriate for children. There is a natural sequence of childhood diseases and acute conditions which prevent children from carrying out their normal activities but may not reflect their health capital or "permanent" health. A useful distinction to make

here is between "permanent" health--one's prospect for life preservation and normal lifetime functioning--and "transitory" health--short-run deviations from one's normal state of health. It is the child's "permanent" health status that we wish to study, and we seek to use health measures which are good indicators of that "permanent" health status.[4]

In some situations a single overall index of permanent health might be desired--to parsimonisouly describe the health status of a population, for example, or to allocate public funds. Infant mortality statistics are frequently used in this way. Health, however, is clearly a multi-dimensional concept. Consequently we use a set of health measures rather than a single index. Analysis of a set of component measures also avoids the essentially arbitrary weighting of the various dimensions of health implied by a health index.[5] Finally, such analysis allows for the possibility that the various aspects of children's health are differentially related to family income and race.

The actual choice of components of children's health status to be examined has been guided by the child health literature,[6] as well as by discussions with public health pediatricians.[7] The measures are listed and described below.

1. The parent's assessment of the child's overall current health, represented by PFGHEALTH. PFGHEALTH is a dichotomous variable indicating whether the parent views the child's health as poor, fair, or good (as opposed to very good).

2. Current height, represented by IHEIGHT. Height is a standard indicator of children's nutritional status [for example, National Center for Health Statistics (1970) and (1975); Seoane and Latham (1971)], and good nutrition is an obvious and natural vehicle for maintaining

children's health. Since it is well known that physical growth rates differ by age and sex, IHEIGHT is computed as the difference between the child's actual height and the mean height for his or her age-sex group divided by the standard deviation of height for that age-sex group.[8]

3. The child's visual acuity, represented by the dichotomous variable ABVIS. ABVIS indicates if the child has abnormal distance vision. All children were examined without their eyeglasses; their uncorrected binocular distance vision is defined as abnormal if it worse than 20/30 [NCHS (1972)].

4. The child's blood pressure, represented by HDBP. HDBP is a dummy variable which indicates if the child's diastolic blood pressure exceeds the 95th percentile for his or her age and sex.

5. Whether or not the child has hayfever or other allergies, represented by the dummy variable ALLEG.

6. An assessment of the child's level of tension, represented by the dummy variable TENS. TENS characterizes children who are rated by their parents as "high strung" or "moderately tense." Both the tension and allergy variables may be regarded as measures of the "new morbidity."

7. The presence of one or more "significant acquired abnormalities" on physical examination of the child, represented by the dummy variable ACABN. These abnormalities include heart disease; neurological, muscular, or joint conditions; and other major diseases.[9]

8. The child's periodontal index, represented by APERI. APERI is a good overall indicator of oral health as well as a correlate of nutrition [Russell (1956)]. Since the periodontal index is known to differ systematically by age and sex, APERI is standardized by age and sex in the same manner as is height. Higher values of APERI denote poorer values of oral health.[10]

9. Excessive absence from school for health reasons during the past six months, represented by the dichotomous variable SCHABS. This variable is taken from information provided by the child's school.[11]

Precise definitions of the above health measures appear in Table 1 along with a notation concerning the source of each variable (medical history, physician's exam, birth certificate, or school form). As is implied in the above definitions, IHEIGHT is a positive correlate of good health and PFGHEALTH, ABVIS, HDBP, ALLEG, TENS, ACABN, APERI, and SCHABS are negative correlates of good health.

III. Race and Income Differences in Health Status in the Cycle II Sample

Table 2 presents the mean levels of these nine health measures for the whole sample as well as by family income and by race.[12] The two family income classes are under $5,000 per annum and $5,000 or more per annum. This income cutoff is selected because it identifies the lowest quartile of the income distribution for the Cycle II sample. For purposes of comparison, Table 2 also includes statistics on infant mortality and the incidence of low birth weight for corresponding income and race classes. Comparable data for childhood mortality are not included because they do not exist.

Health differences by race are significant (at the 5 percent level in a two-tailed test) for three of the nine measures: PFGHEALTH, ALLEG, and TENS. Black children are in worse health than white children according to their parents' assessment of their overall current health (also according to HDBP and ACABN, although for these two the race differences are not statistically significant). Black children are in <u>better</u> health, however, in that they exhibit a lower incidence of allergies and tension

TABLE 1
Definitions of Health Measures

Variable Name	Definition	Source*
PFGHEALTH	Dummy variable that equals one if parental assessment of child's health is poor, fair, or good and zero if assessment is very good	1
IHEIGHT	Height, standardized by the mean and standard deviation of one-year age-sex cohorts	3
ABVIS	Dummy variable that equals one if uncorrected binocular distance vision is abnormal	3
HDBP	Dummy variable that equals one if the child's diastolic blood pressure is above the 95th percentile for his age and sex	3
ALLEG	Dummy variable that equals one if the child has had hayfever or any other kind of allergy	1
TENS	Dummy variable that equals one if the parent rates the child as high strung or moderately tense	1
ACABN	Dummy variable that equals one if the physician finds a "significant" acquired abnormality in examining the child (other than an abnormality resulting from an accident or injury)	3
APERI	Peridontal index, standardized by the mean and standard deviation of one-year age-sex cohorts	3
SCHABS	Dummy variable that equals one if child has been excessively absent from school for health reasons during the past six months	4

*The sources are 1 = medical history form completed by parent, 2 = birth certificate, 3 = physical examination, 4 = school form completed by teacher or other school official.

TABLE 2
Mean Values of Health Measures in Cycle II Sample by Family Income and Race, and Infant Mortality and Birth Weight by Family Income and Race

Cycle II Health Measure	Cycle II Working Sample[a] (n = 4777)	Blacks (n = 581)	Whites (n = 4196)	"z" Statistic for White-Black Difference[b]	Income < $5,000 p.a. (n = 1645)	Income ≥ $5,000 p.a. (n = 3132)	"z" Statistic for High-Low Income Difference[b]
PFGHEALTH	.4706	.6127	.4509	-7.32	.5939	.4058	-12.38
IHEIGHT[c]	.0258	.0900	.0169	1.68	-.1177	.1012	7.35
ABVIS	.1160	.1136	.1163	.19	.0954	.1268	3.22
HDBP	.0557	.0671	.0541	-1.28	.0614	.0527	-1.25
ALLEG	.1478	.0878	.1561	4.35	.1015	.1721	6.53
TENS	.4593	.3373	.4762	6.30	.4304	.4745	2.91
ACABN	.0373	.0430	.0365	-.77	.0389	.0364	-.33
APERI[c]	-.0392	-.0736	-.0344	1.19	.0711	-.0971	-7.48
SCHABS[d]	.0445	.0422	.0449	.30	.0483	.0426	-.91

Other Health Measures	Total Population 1963-65	Black Population 1963-65	White Population 1963-65	Population with Income < $5,000 p.a. 1963-65	Population with Income ≥ $5,000 p.a. 1963-65
Infant mortality[e]	23.0	39.5	20.8	28.4	19.1
Percent of births with weight < 2,500 grams	7.87	14.01	7.01	8.89	7.05

Footnotes to TABLE 2

[a]For description of working sample, see footnote 12.

[b]The critical value for "z" at the 5 percent level of significance in a two-tailed test is 1.96.

[c]The mean of this variable is not zero because standardization was done using the means and standard deviations for the entire Cycle II sample rather than for our working subsample.

[d]Means serived from subsample of working sample for which a school report was available (n = 4333 rather than 4777).

[e]Deaths per 1,000 live legitimate births. Source: Infant Mortality Rates: Socioeconomic Factors, N.C.H.S. Series 22, No. 14, Table 3, p. 14, U.S. 1964-66.

(favorable differences are also apparent for IHEIGHT, ABVIS, APERI, and SCHABS, although these are not statistically significant). Race comparisons of children's health based on these nine measures, therefore, clearly yield a much less uniform impression than is drawn from inspection of data on infant mortality and low birth weight. Rather than exhibiting the dramatically large health deficits of black infants, older black children are in some ways worse off but also in some ways better off than their white counterparts.

This difference between the relative health profile of blacks in infancy and in midchildhood is not simply a result of differences in data sources or differences between sample data and population data. Available data on the Cycle II children during their infancy (birth weight, the incidence of congenital abnormalities, and the parents' retrospective assessment of the infant's health) indicates that the black children had significantly poorer health in infancy. The difference in relative profiles, however, is in part a result of the difference in types of measures studied. Our measures pertain to specific health characteristics rather than to mortality. When mortality data for 5 to 14 year-old children in a corresponding year (1964) are examined, they do show that blacks have higher death rates. Childhood mortality is about 47 percent higher for blacks (although this ratio reduces to 37 percent when accidents, suicides and homocides are excluded).[13] Even so, this ratio is much smaller than the corresponding difference in infant mortality. Thus, in the midchildhood years, black-white health differences--no matter how they are measured--are greatly attenuated as compared to such differences in infancy.

Significant income differences are found for six of the nine health measures. As in the case of race differences, the nature of these differences is not uniform. Children from higher income families have significantly better health as measured by PFGHEALTH, IHEIGHT, and APERI (also HDBP, ACABN, and SCHABS although for these three the income differences are not significant). On the other hand, they have significantly poorer health as measured by ABVIS, ALLEG, and TENS.[14] Again, the overall impression conveyed by these data is less clear-cut than that obtained from examining income differences in infant mortality and birth weight: in some cases children from low income families do have poorer health than those from higher income families, but with respect to measures that reflect the "new morbidity" it is the children from low income families who appear to be in better health.[15]

IV. Decomposition of Observed Race and Income Differences

We have documented the fact that race and income differences in health status in Cycle II sample children are much less sharp than are corresponding differences in measures of infant health. Nevertheless, some differences still do exist. To what extent are these uniquely associated with income and race and to what extent can they be attributed to correlated socioeconomic factors?

A. Race Differences

It is well known that race and income are themselves highly correlated. Thus it is not surprising that the three health measures displaying significant race differences also exhibit significant income differences (see Table 2). An obvious first step, therefore, is to try to determine if these observed race differences are really just a result of

differences in income. To do so we obtain mean values for the nine health measures when the Cycle II sample is cross-classified by both income and race (Table 3). Among low income families a significant race difference remains for only one of the three variables, tension. In addition, for IHEIGHT and APERI the race difference is now significant where it was not before. For all three of these measures, however, black children are found to be in better health than white children from families of comparable low income levels. In high income families, significant race differences still exist for PFGHEALTH, ALLEG, and TENS, with black children in worse health according to PFGHEALTH and in better health according to TENS and ALLEG. Thus, within income classes significant health differences still exist between black and white children. As before, these differences do not uniformly favor children of either race, but most of the significant differences show blacks to be better off.[16]

A further way of investigating the nature of race differences in children's health is to look at residual race differences after a much larger list of socioeconomic variables is held constant. This is done using multiple regression analysis. The dependent variables in the regression equations are the nine health status measures. For the explanatory variables we use a set suggested by the economic model of family investment in children's health described in Edwards and Grossman (1978). Included are family income, parents' educational attainment, whether the child's father lives with the family, whether the child is a twin or is a first-born, whether a foreign language is spoken in the home, an indication of the region of residence and size of city of residence, and the sex of the child (the latter is included only for health

TABLE 3

Gross and Net Race Differences in Children's Health Status

Health Measure	Gross Difference (Black Mean-White Mean)	Low Income Families (≤ $5,000 p.a.)			High Income Families (≥ $5,000 p.a.)			Net Difference[a]
		Black Mean (n = 418)	White Mean (n = 1,227)	Difference	Black Mean (n = 163)	White Mean (n = 2,969)	Difference	
PFGHEALTH[c]	.162**	.612	.589	.023	.614	.394	.220**	-.357**
IHEIGHT[c]	.073*	.068	-.181	.249**	.146	.099	.047	.657**
ABVIS[b]	-.002	.108	.091	.017	.129	.127	.002	.009
HDBP[b]	.013	.069	.056	.013	.061	.052	.009	-.003
ALLEG[c]	-.068**	.084	.108	-.024	.098	.176	-.078**	.227**
TENS[b]	-.139**	.354	.456	-.102**	.295	.484	-.189**	-.153**
ACABN[b]	.007	.046	.037	.009	.037	.036	.001	.018*
APERI[c]	-.039	-.063	.117	-.180**	-.101	-.097	-.004	-.443**
SCHABS[b]	-.003	.045	.049	-.004	.034	.043	-.009	-.005

Footnotes to TABLE 3

*,**Significant race differences in means at the 10 percent and 5 percent levels of significance, respectively.

[a]See text for description.

[b]Net difference equals regression coefficient of a race dummy variable (1 = black) from a pooled regression of black and white children that holds constant all other independent variables.

[c]Net difference equals difference in intercepts between black and white regressions with all independent variables held constant.

measures that are not standardized by sex). These variables are defined in detail in Chapter 2.

Race differences in children's health net of differences in this set of socioeconomic variables are presented in the last column of Table 3.[17] In general, the net differences are not smaller than the gross differences. In fact, the net differences are significant for five rather than three of the nine variables, with blacks exhibiting better health for four of the five. This picture changes only slightly if we include ACABN for which blacks exhibit poorer health, but only at the 10 percent level of significance. In sum, when the above set of socioeconomic variables is held constant, being black is associated with significantly better health for children when their health is measured according to their parents' assessment, their height, their tension level, and their periodontal index, and with significantly worse health according to the prevalence of allergies and possibly, acquired abnormalities. In addition, comparison of these net differences with the gross differences (for all incomes or within income classes) reveals two changes in the health rankings of black and white children: the parental assessment measure now favors blacks and the allergy measure now favors whites.

What conclusions do we draw from the various statistics in Table 3? First, that race differences in children's health do not disappear when income and various other socioeconomic variables are held constant; if anything, they increase. Second, these differences do not obviously favor children of either race. As to the reasons for these differences, one can speculate about the roles of such factors as differential genetic endowments (in the case of height or the periodontal index, for example) and differences in tastes or in life styles.

B. Income Differences

Race differences in children's health did not disappear when various socioeconomic factors (including income) were held constant; does the same conclusion hold for income-related differences? Put differently, to what extent do the gross differences in health status between income classes disappear when additional socioeconomic factors are held constant? To answer this question, we use the same type of multiple regression analysis described earlier. We simplify the analysis, however, by restricting the decomposition of gross income differences to the white sample only.[18]

Gross and net income differences for the nine health measures are presented in Table 4. The "net" income differences are computed similarly to the corresponding race differences[19] and should be interpreted as the difference in mean health status between the two income classes if all of the socioeconomic variables (other than income, of course) took the same values in both classes. The number of health status measures for which significant income differences exist is reduced from five to three when related socioeconomic factors are held constant. In all three cases low income is associated with significantly poorer health. (In the case of ALLEG, low income is associated with better health, but relationship is significant only at the 10 percent level.) In addition, the magnitude of these differences is substantially reduced, with the net differences being typically less than half of the gross differences (see last column of table). For example, on the basis of the gross difference, about 20 percent more of the high income parents than of the low income parents assessed their childrens' health as very good. When related socioeconomic factors are held constant, this difference is reduced to only 7 percent. Thus, although income differences in health status still

TABLE 4

Gross and Net Income Differences in the Health Status of White Children[a]

Health Measure	High Income Mean	Low Income Mean	Gross Income Difference[b]	Net Income Difference[c]	Ratio of Net to Gross Income Difference
PFGHEALTH	.394	.589	-.195**	-.072**	.37
IHEIGHT	.099	-.181	.280**	.117**	.42
ABVIS	.127	.091	.036**	.020	.56
HDBP	.052	.056	-.004	-.001	.25
ALLEG	.176	.108	.068**	.028*	.41
TENS	.484	.456	.028*	-.009	d
ACABN	.036	.037	-.001	-.004	2.00
APERI	-.097	.117	-.214**	-.077**	.36
SCHABS	.043	.049	-.006	-.001	.17

[a]There are 2,969 white children in the high-income ($\geq$ \$5,000 p.a.) sample and 1,227 white children in the low-income ($\leq$ \$5,000 p.a.) sample.

[b]This is computed as the mean in the high income sample minus the mean in the low-income sample.

[c]This is the coefficient of a dummy income variable (high-income children have value of one) in a multiple regression which includes variables listed in text.

[d]Not computed because of change in sign.

*,**Significant income differences at the 10 percent and 5 percent levels of significance, respectively.

do exist, their magnitudes are greatly diminished when related factors are held constant. The main conclusion to be drawn is clear: gross income differences in health greatly overstate the true relationship between family income and health.

Further insight about the nature of gross income differentials in health status is obtained by studying the precise role of the explanatory socioeconomic variables. Table 5 presents calculations which illustrate how the gross income differences are decomposed among the various explanatory factors for the six health variables exhibiting significant gross income differences at the 5 or 10 percent level. The procedure is simply to multiply the coefficients of these explanatory variables by the differences in their mean values in the high and low family income samples of children.[20] Several results in Table 5 are noteworthy. First, almost all of the differences in the six health measures between the high and low income subsamples can be accounted for by differences in the independent variables that we have included in our equations. Second, a detailed examination of the decomposition indicates that for four of the six measures (PPGHEALTH, IHEIGHT, ALLEG, and APERI), differences in parents' average schooling between high and low income families account for as much or more of the gross differences as does income itself. For the other two measures, ABVIS and TENS, no single component accounts for a large part of the gross differences, but rather many components play small roles.

One interesting implication emerges from this decomposition. It does not appear that it is their greater access to medical care that causes higher income families to have, on balance, healthier children. Rather, it is their enhanced knowledge--as reflected by their higher

TABLE 5

Components of the Difference in PFGHEALTH, IHEIGHT, ABVIS, ALLEG, TENS and APERI Between White Children from High and Low Income Families

Component	PFGHEALTH	IHEIGHT	ABVIS	ALLEG	TENS	APERI
Family income	-.072	.117	.020	.028	-.009	-.077
Parents schooling	-.089	.126	.002	.045	.005	-.145
Other family characteristics[a]	.000	.009	.004	.004	.012	-.011
Characteristics of child[b]	-.013	.008	.000	.001	.007	.001
Region	-.024	.010	.007	-.005	-.002	.020
City size	-.006	.009	.004	-.004	.016	-.003
Total = Predicted Gross Difference	-.205	.279	.037	.069	.029	-.215
Actual Gross Difference	-.195	.280	.036	.068	.028	-.214

[a]These include whether or not a foreign language is spoken in the home and whether or not the father is absent from the home.

[b]These include whether or not the child is a first-born or a twin, and his or her sex. The latter is not included for IHEIGHT and APERI, both of which are standardized by sex.

levels of educational attainment--about what it takes to bring up healthier children. Thus, the usual policy recommendations of income redistribution or redistribution of medical resources would not be supported by the evidence in Table 5.

Our findings with respect to race and income differences may be contrasted with those in a recent study of infant mortality by Gortmaker (1977). He attempts to determine what portion of the large income and race differences in infant mortality can be explained by differences in parents' educational attainment, mother's age, the child's birth order, and the previous pregnancy experience of the mother. He finds that poverty families and black families still display a much higher incidence of infant mortality even when these factors are controlled for.

V. Conclusions

In our analysis of data for a national sample of children in the mid-childhood years, we have reported differences both according to race and according to income, but these differences by no means overwhelmingly favor the white or high-income children. With respect to differences by race, whether or not they are adjusted for differences in associated socioeconomic factors, black children in some cases are in better health than their white counterparts. When income differences are the subject of analysis, the high income children do appear to be in better health according to most measures, but their advantage is greatly diminished when one controls for related socioeconomic factors like parents' educational attainment. Even so, for measures relating to the new morbidity children from higher income families appear to be in worse health. It is possible to argue that the latter finding is a statistical artifact because health problems like

allergies and excessive tension are comparatively subtle and are detected only in a setting (i.e. in a high income environment) where other types of health problems are minimized. But our finding that the incidence of the new morbidity is greater in children from high as opposed to low income families is consistent with the well known positive relationship between income and morbidity and mortality rates observed for adults in the United States [for example, Auster et al. (1969) and Grossman (1975)].

A more general conclusion to be drawn from our results is that income and race differences in infant mortality provide a poor, and even misleading description of income and race differences in the health of older children. It may be appropriate to use infant mortality statistics in broad across-country comparisons of the health status of various populations, but these statistics should not be used to compare the health status of various groups of older children within the United States.

In addition, our findings highlight the necessity of explicitly recognizing the multidimensional nature of health. For example, poor and black children are in worse health when traditional health measures are used, but they tend to be in better health when aspects of the new morbidity are under study. Such fine distinctions are hidden when a single index like infant or childhood mortality is used. These results underscore the importance of treating children's health status as multidimensional and illustrate how the use of a single health index could lead to erroneous conclusions about health status and its relation to income and race.

Two important and as yet unanswered questions are raised by the findings in this study. The first relates to the race differences in

children's health that exist even after a host of socioeconomic factors are held constant. What are the causes of these differences, and what do they mean? The second arises out of the divergence between infant mortality statistics and our mid-childhood health measures. Why are the income and race differences in infant mortality so striking while the corresponding differences in mid-childhood morbidity are not? This divergence is not simply a result of the type of measure (mortality versus morbidity); differences in the mortality of older children are also less striking. One possible explanation is that the black children whose health prospects are the worst die in infancy so that those who survive to mid-childhood do not differ as greatly from surviving white children. Whatever environmental factors at work would seem to have a much larger impact in infancy than later in childhood. If this is so, we need to know why. The answers to these questions are clearly pertinent to the conduct of public policy towards the welfare of children.

The primary focus of this study has been to clarify commonly held notions about income and race differences in children's health. In doing so, we have generated results that are different from what one would expect and also, perhaps, from what one would like to find. If we had found that race differences disappeared when income and other socioeconomic factors were held constant, and that large income differences remained after other socioeconomic characteristics were held constant, simple and clear-cut policy implications would follow. In that case an income redistribution or a redistribution of market health inputs (for example, medical care, a nutritious diet, a clean physical environment), would lead to an improvement in the health status of poor

white and black children. Our actual findings--that race differences in health do not disappear when income and other socioeconomic factors are held constant, and that income differences in health do greatly diminish when other socioeconomic factors are held constant--do not suggest simple remedies for eradicating existing health differences. What comes to mind is some type of educational program tailored to reach precisely those families who need it. Whether such a program can be successful is questionable, but then, if there is anything that we have learned over the past 15 years of government-sponsored attempts at social change, it is that simple solutions to social problems are not readily uncovered.

FOOTNOTES

[1]A full description of the sample, the sampling technique, and the data collection is presented in NCHS (1967). The one deficiency of this sample from the point of view of studying children's health is the exclusion of children in institutions. To the extent that these children are more likely to have serious and disabling physical conditions, the reported incidence of certain conditions will be lower in our sample than in the entire population of children. In addition, if the probability of the institutionalization of a child with a given condition depends on income and race, our results will incorporate unknown biases. The number of institutionalized children is small, however, at about four-tenths of a percent of all children aged 5 through 13 years. [This is the proportion of 5-13 year-olds living in "group quarters" in 1970 according to the U.S. Bureau of the Census (1973), Tables 52 and 205. The corresponding percentages by race are .38 percent for whites and .7 percent for blacks.]

[2]See, for example Sullivan (1966), Berg (1973), and, more recently, Ware (1976).

[3]See Grossman (1972), p. 58. This definition is also similar to that proposed in Torrance (1976).

[4]There is, of course, a positive relationship between "permanent" and "transitory" health status in the sense that a child with low health capital is more likely to contract some acute conditions and to have them for a more extended time period. For example, Birch and Gussow (1979) discuss how nutrition (which is clearly a determinant of "permanent" health status) and disease are intimately related.

[5]In earlier work some attempts were made to condense the health information using principal component analysis. The analysis yielded almost as many equally weighted components as there were initial health measures.

[6]The studies we consulted are: Wallace (1962); Mechanic (1964); Mindlin and Lobach (1971); Talbot, et al. (1971); Kaplan et al. (1972); Hu (1973); Starfield (1977); Kessner (1974); Haggerty et al. (1975); and Inman (1976).

[7]The following physicians gave us helpful advice: John McNamara, M.D., then Assistant Professor of Public Health and Pediatrics at Columbia University School of Public Health and Associate Commissioner in the New York City Department of Health; Roy Brown, M.D., Associate Professor of Community Medicine and Pediatrics at the Mount Sinai School of Medicine of the City University of New York; Thomas Travers, D.D.S.,

Director of Ambulatory Care in the New York City Department of Health; and Puth T. Gross, Professor of Pediatrics and Director of Ambulatory Pediatrics, Stanford University Medical Center, Stanford, California.

[8]If the actual height of each age-sex group is normally distributed, IHEIGHT can be translated directly into the child's height percentile.

[9]In defining ACABN, we exclude abnormalities resulting from accidents or injuries because these are more likely to reflect transitory rather than permanent health variations.

[10]The periodontal index suffers from the defect that it is subject to intra-rater and inter-rater variability. We have experimented with a somewhat more objective measure of oral health, the number of decayed permanent and primary teeth adjusted for age and sex, and have obtained results similar to those for the periodontal index. Compared to the number of decayed teeth, the periodontal index reflects more serious oral health problems.

[11]There is no school form for approximately 500 children in the Cycle II data set. Since excessive absence due to illness is the only variable taken from the school form, children without the school form are eliminated from our working sample only when school absenteeism is examined.

[12]These means are computed using only 4,777 of the 7,119 observations because our working sample only includes children who lived either with both of their parents or with their mothers only (no step-parents, foster parents, grandparents, etc.). In addition, the 72 children who turned

12 years old after having been chosen for the Cycle II survey are excluded as well as those children for whom there were missing data on either these health measures or on the socioeconomic variables used in the next section. Comparison of these means with corresponding means for the entire Cycle II sample reveals that there is very little difference between the full sample and our working subsample.

[13]See U.S. Department of Health, Education and Welfare (1966), Tables 1-10.

[14]One might object to the use of subjective measures like PFGHEALTH, TENS, and ALLEG to compare the health of different income and racial groups because such measures may be subject to systematic reporting bias. Specifically, more educated parents (who are also more likely to be white and to be in the upper income class) may be more likely to observe and report their children's problems with allergies and tension. If so, white children and children in the upper income class will be reported to exhibit a higher incidence of these problems even if they do not. This bias may be a problem in this section, where only gross comparisons are made, but it is not in the next section, in which parents' education and many other family characteristics are controlled for.

[15]Income differences were also computed for an all-white sample with virtually identical results (see Table 4). Thus, these reported income differences in health are not the result of the higher proportion of blacks in poor families.

[16]These results can again be contrasted with comparable data for the two infant health measures discussed earlier. Even within income classes,

race differences in infant mortality and in the incidence of low birth weight remain large and consistently favor whites:

	Low Income (≤ $5,000 p.a.)		High Income (≥ $5,000 p.a.)	
	Blacks	Whites	Black	Whites
Infant mortality	43.9	24.2	23.6	18.6
Incidence of low birth weight	13.2	7.6	17.1	6.6

(Source and definitions of health measures are the same as in Table 2).

[17]Net differences are computed in two different ways depending on whether or not there are significant race differences in slope coefficients in the underlying health equations. If there are no significant differences in the slope coefficients by race (as in the case for the dependent variables HDBP, ABVIS, TENS, ACABN, and SCHABS), the "net" race difference is represented by the regression coefficient of a race dummy variable (black = 1) from a pooled regression of black and white children that holds constant all other dependent variables. In this case, the "net" difference is that portion of the gross difference that remains after holding constant differences in the mean values of the explanatory variables. When there are significant race differences in the slope coefficients (as in the case of the dependent variables APERI, IHEIGHT, PFGHEALTH, and ALLEG), the net differences is computed as the difference in intercepts from race-specific regression estimates with the set of explanatory variables held constant. In this case the net difference represents that portion of the gross race difference that

remains after allowing for race differences in both means and in slope coefficients.

[18]The list of health measures for which there are significant income differences is the same for the white sample as it is for the full black and white sample reported on in Table 2.

[19]In no cases were there significant differences in the slope coefficients in the two income classes.

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Chapter 4

ADOLESCENT HEALTH, FAMILY BACKGROUND, AND PREVENTIVE MEDICAL CARE

This study investigates the health of white adolescents, focusing particularly on the roles of family background and preventive medical care. This emphasis is motivated in part by our desire to study adolescent health in the context of the nature-nurture controversy. Despite the existence of a massive literature on the relative importance of heredity (nature) and the home and school environment (nurture) in the determination of cognitive development,[1] the corresponding issue has not been directly addressed by researchers in child and adolescent health. This is partly because much of the health research is limited either to poverty or to minority populations (Hu 1973; Kessner 1974; Inman 1976; Dutton 1978; Ditton and Silber 1979), and partly because researchers who use representative samples do not adopt the multivariate context necessary for distinguishing between genetic and environmental influences (Douglas 1951; Douglas and Bloomfield 1958; Kellmer-Pringle, Butler, and Davie 1966; Haggerty, Roghmann, and Pless 1975; Zimmer 1978). Our research uses multivariate statistical techniques to provide some evidence of the degree to which nurture--that is, the family and local environment--acts in determining the health levels of a representative sample of white adolescents.

One aspect of the adolescent's environment, medical care, has been recognized as the logical vehicle for public policy aimed at improving adolescent health. For example, Newberger, Newberger, and Richmond (1976), Keniston and the Carngeie Council on Children (1977), and Marmor (1977) all have proposed that national health insurance should provide coverage of

for choosing these indicators is that they represent health problems that are capable of being affected by family decisions concerning diet and other forms of at-home health care, as well as by pediatric and dental care. This is in contrast to many adolescent health problems that are either self-limiting, such as morbidity from acute conditions, or irreversible, such as congenital abnormalities of the neurological system.

To analyze these health problems we use data from Cycle III of the U.S. Health Examination Survey (HES), an exceptional source of information about a national sample of 6,768 noninstitutionalized youths aged 12 to 17 years in the 1966-70 period.[2] The data comprise complete medical histories of each youth provided by the parent, information on family socioeconomic characteristics, and birth certificate information. Most important, there are objective measures of health from detailed physical examinations given to the youths by pediatricians and dentists employed by the Public Health Service. These data are supplemented by two medical resource inputs specific to the youth's county of residence (the number of pediatricians per capita and the number of dentists per capita) and information on the presence of controlled or natural fluorides in the water supply system that services the youth's community. The last piece of information enables us to evaluate the impact of a collective, as opposed to an individual, preventive dental practice.

These data are used to estimate two types of relations: a health production function and a derived demand function for preventive care. The resulting estimates permit us to answer the following four questions. What is the size of the home environmental effect on adolescent oral and physical (obesity, anemia, corrected distance vision) health outcomes?

prenatal care, pediatric care, and dental care. Bills with this aim have been introduced in Congress by Senator Jacob K. Javits and Congressman James H. Scheuer, both of New York. To cite another illustration, recently-enacted Federal legislation has attempted to increase the availability of pediatricians and dentists in medically underserved areas to expand the use of preventive care in such areas. The Emergency Health Personnel Act of 1970 (PL 91-623) created the National Health Service Corps., whose members are assigned to health manpower shortage areas. The Health Professions Assistance Act of 1976 (PL 94-484) encourages new graduates of medical and dental schools to locate in urban ghettos and rural regions by forgiving their medical education loan obligations. Further, the Health Maintenance Organization Act of 1974 (PL 93-222) gives priority for developmental funding of HMOs in medically deprived areas. One objective of our research is to provide estimates of the potential payoffs to national health insurance and medical manpower policies directed at improving youths' health.

The specific health indicators we study are oral health, obesity, anemia, and corrected distance vision. These four are chosen not only because they represent health problems that create discomfort for the teenager, but more importantly, because they may be good predictors of subsequent adult health. Indeed, they all partly reflect poor health habits that are likely to persist into adulthood. With the growing evidence that adults' choice of life styles and health behaviors can have important impacts on their health (Breslow and Klein 1971; Fuchs 1974a, 1974b; Grossman 1975; Manheim 1975), it is natural to look into adolescence to understand the formation of these habits. A second motivation

How important is the home environment as a determinant of the demand for preventive dental and pediatric care? How large are the effects of dentists, preventive dental care, and fluoridation on oral health outcomes? How large are the effects of pediatricians and preventive pediatric care on physical health outcomes? In addressing the last two questions, we recognize explicitly the common-sense proposition that an increase in a community's physician or dental manpower will not increase health outcomes unless it encourages more utilization of medical care services. Previous empirical work on the impact of physicians or dentists on health has not taken account of this restriction (for example, Newhouse and Friedlander 1977).

Our findings indicate first, that family characteristics do have a significant impact on adolescent health and second, that preventive care is an important vehicle for this impact in the case of dental health but not in the case of the three physical health measures. Similarly, the greater availability of dentists has a positive impact on dental health, but greater availability of pediatricians does not alter the physical health measures. On the basis of these results we predict that government efforts to improve the dental health of adolescents with policies to lower the cost of dental care or increase the availability of dentists are much more likely to be successful than similar policies directed at improving their physical health.

I. Analytical Framework

Previously, in Charter 2, we argued that offsprings' health can be examined fruitfully within the context of the economic models of fertility developed by Becker and Lewis (1973), Willis (1973), and

Ben Porath and Welch (1976). In these models the parents' utility function depends on their own consumption, their family size, and the "quality" of each child. Child "quality" refers to those characteristics of the child that generate utility for the parents: his health, sex, wealth, social adjustment, intellectual development, etc. Therefore, when parents choose their optimal family composition, they choose not only how many children they will have but also what portion of the family's resources will be devoted to each child. This choice is made in the usual way: parents choose the number and quality of children, as well as of other consumption goods, so as to maximize their utility subject to the constraints imposed by their wealth (their potential earned and non-earned income) and the various prices they face. In the case of children, there is a further constraint in the form of children's genetic endowments which in part determine their quality. Genetic endowments act as a constraint because they are largely outside of the family's control.

The prices of children and of the various components of their quality are determined by a fundamental insight embedded in the household production function approach to consumer behavior: consumers produce their basic objects of choice with inputs of goods and services purchased in the market and their own time (Becker 1965). This insight is of particular relevance in dealing with children and their health because parents obviously do not buy these objects of choice directly in the market; both a child's home environment and his genetic endowment are important determinants of his ultimate health level. Therefore, the price of health depends on the cost of the parents' or other caretakers' time, and the prices of medical care, nutrition, and any other purchased inputs used to

improve children's health. It also depends on the number of children in the family because the more children there are in the family, the more costly it is to raise their average health level. In addition, to the extent that there are systematic differences in the ability of families to produce children's health with given inputs, these differences in efficiency are also relevant. For example, more educated parents are more likely to be able to follow doctors' instructions, to have general information about nutrition, and to be willing and able to acquire medical information from published materials. Consequently, one would expect more educated parents to be more efficient in producing healthy children.

Given these considerations, the following factors are expected to influence children's health levels: the child's exogenous (genetic) health endowment, family wealth, parents' wage rates, family size, parents' educational attainment and other measures of their efficiency in household production, and the direct and indirect costs of medical care and other market health inputs (vitamins, sanitation, etc.).[3] (The indirect costs of medical care are generated by the time spent in traveling, waiting, and obtaining information about this care.[4]) The relationship between the child's ultimate health and this set of factors may be termed a demand function for the output of health. In this demand function a positive association between children's health and family wealth is predicted (assuming that child health is a normal good). Similarly, a positive association is expected between both parents' education and children's endowed health status and children's ultimate health status. Negative associations would be anticipated between all of the prices of health inputs and children's health, and between family size and children's health. Parents' wage rates may have negative or positive effects

on children's health levels depending on whether the household production of children's health is more or less time intensive than the production of other aspects of child quality and/or other types of parents' consumption commodities. In this framework a child's health is treated as a single datum--his permanent health measured, say, at the beginning of adulthood or as an average over his childhood and adolescence. This type of model is not formulated to explain variations in health over childhood or to examine the child's contribution to his own health.[5]

The above model provides a useful setting within which to view adolescent health, but empirical estimation of the resultant "demand for health" function would not yield answers to the questions posed in the introduction. Such estimates would only yield information about the total impact of family characteristics or medical input prices on children's health. To determine the effect of preventive care on health we need estimates of the health production function. Similarly, to determine whether families with specific characteristics are more efficient at producing healthy children also requires estimates of this production function. Alternatively, to assess the role of family characteristics in determining the amount of preventive care received by adolescents, an estimate of a derived demand function for medical care is needed. Finally, a computation of the impact of health manpower availability on adolescent health requires not only the above functions but also a set of market demand and supply for health manpower functions. In the latter case, we employ a simplified approach which yields rough estimates of these manpower availability effects on health.

A. The Health Production Function

A simple, linear health production function[6] is represented by

$$(1) \qquad H = \beta_0 + \beta_1 E + \beta_2 G + \beta_3 M + \beta_4 X + \beta_5 R + u_1 \quad .$$

Here H is a health measure, E is a vector of family efficiency characteristics, G is a vector of the adolescent's endowed health characteristics, M is a medical or dental care input, X is a vector of other family inputs (nutrition, parents' time, etc.), R is a vector of relevant regional characteristics (city size, region of the country, and whether or not the water supply is fluoridated), and u_1 is a random error term with the usual properties.

The health production function actually estimated in Section III does not correspond exactly to equation (1) because of inadequate data. First, data on the amount of "other" inputs (X) are not available. Therefore, we include the following proxy measures for X: family income, family size, and the mother's labor force status. Family income is positively related and family size is negatively related to nutrition and other unmeasured market health inputs. Family size and mother's labor force status are proxies for the amount of time the mother spends with each of her offspring. Women who work full-time or part-time in the labor market and women with many offspring have less time to spend with each one. In addition, our data do not include good information about curative care. Consequently, M represents only preventive care. This is not a serious deficiency because we have chosen health measures for which the impact of preventive care (with the associated remedial treatment) is

relatively large. (By focusing on health problems for which the medical input is primarily preventive, we also avoid the necessity of modeling the simultaneous determination of health levels and curative care utilization.)

B. The Derived Demand for Preventive Care

The derived demand function for medical care depends on the same set of variables as the demand function for health:

$$M = \gamma_0 + \gamma_1 F + \gamma_2 G + \gamma_3 P + \gamma_4 R + u_2 \quad . \tag{2}$$

F represents family income, education, family size, and other family characteristics affecting either the demand for health or the family's efficiency in producing healthy children; G and R are the same as in equation (1); P represents a vector of relevant direct and indirect input prices (wage rates, the cost of a doctor or dental visit, etc.); and u_2 is the usual random error term.

We cannot estimate this derived demand curve exactly as stated because data on P are not available. Inclusion of variables representing the mother's labor force status helps control for variations in the mother's wate rate. Other input prices are partially controlled for by the region and city-size variables in R. Finally, physician or dentist availability measures are included to represent differences in the direct and indirect costs of medical or dental care.[7] Thus, rather than equation (2), we estimate the following:

$$M = \alpha_0 + \alpha_1 F + \alpha_2 G + \alpha_3 D + \alpha_4 R + u_3 \quad . \tag{3}$$

where the vector F now includes the mother's labor force status and D

represents the number of pediatricians or dentists per capita in the adolescent's county of residence.

C. The Role of Health Manpower Availability

It is the inclusion of manpower availability measures in the derived demand for preventive care functions that permits us to obtain a rough assessment of the impact of health manpower on the demand for preventive care, and consequently, on adolescent health. Only a rough assessment is possible because to get precise estimates it is necessary to have, first, data on the direct and indirect costs of medical care and second, measures of the price elasticity of supply of physicians or dentists. Good estimates of the supply elasticities do not exist, and it is almost impossible to measure all of the indirect costs of medical care. Although data on direct costs do exist, they are not usually found in conjunction with the detailed health and family background data used here. Thus, our estimate of the impact of health manpower on health is the best that can be obtained given the limitations of existing data sets. The coefficients of the health manpower variables in the derived demand equations embody both the relationship between health manpower availability and direct and indirect medical care prices, and the relationship between medical care prices and the demand for preventive care.

Implicit in the above discussion is the assumption that an increase in a community's health manpower will not improve the health of adolescents unless it encourages a greater utilization of preventive care services. This assumption is explicitly incorporated in equations (1) and

(3): D is assumed to have no direct effect on health in equation (1) but alters health only via its impact on M in equation (3). Substituting equation (3) into equation (1) yields estimates of the total impact of doctor or dentist availability on health:

$$(4) \qquad H = \beta_0 + \beta_1 E + \beta_3 \alpha_1 F + (\beta_2 + \beta_3 \alpha_2) G + \beta_3 \alpha_3 D$$

$$\beta_4 X + (\beta_5 + \beta_3 \alpha_4) R + u_1 + \beta_3 u_3 \quad .$$

The total impact of pediatrician or dentist availability on health is given by $\beta_3 \alpha_3$. Note that an estimate of the total impact computed from individual estimates of α_3 and β_3 differs from that obtained from direct estimation of equation (4) because the latter does not incorporate the restriction that D does not appear in equation (1).[8]

D. The Role of Family Background Variables

To the extent that there are family background variables common to both the set E and the set F (parents educational attainment is a good example of one), the substitution in equation (4) provides an additional insight. Parents' education is clearly seen to have two effects on adolescent health: a direct or "efficiency" effect given by β_1 and an indirect or "allocative" effect given by $\beta_3 \alpha_1$. The latter refers to the ability of parents with greater schooling levels to select a better input mix in the production function.[9]

II. Empirical Implementation

Equations (1) and (3) are estimated using Cycle III data for white adolescents who live with either both of their parents or with their mothers only. Black adolescents are excluded from the empirical analysis. Preliminary results revealed significant race differences in slope coefficients so that pooling blacks and whites for estimation was inappropriate. Separate estimates for black adolescents are not presented because the black sample is too small to allow for reliable coefficient estimates. Observations are also deleted if there are missing data. The final sample size is 4,121. Table 1 contains definitions, means, and standard deviations of all of the dependent and independent variables. It also contains a notation concerning the source of each variable.

A. Measurement of Adolescent Health

In the introduction to this chapter, we expressed an intention to study physiological measures of adolescent health that (1) reflect detrimental health behaviors or life styles that may persist and create more serious problems in adulthood and (2) relate to problems that can be modified by endogenous inputs in the health production function such as proper diet, parents' time, and especially preventive medical care.[10] Based on these criteria, we focus on two correlates of poor oral health: the periodontal index and the number of decayed permanent teeth; and on three correlates of poor physical health: obesity, abnormal corrected distance vision, and anemia as reflected by low hematocrit levels. All five measures clearly relate to conditions that can carry on into adulthood, and all can be modified by appropriate care. Dental care provided

TABLE 1

Definitions of Variables

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
A. Health Measures				
APERI[c]	-.114	.857	Periodontal index, standardized by the mean and standard deviation of one-year age-sex cohorts	2
IDECAY[c]	-.146	.839	Number of decayed permanent teeth, standardized by the mean and standard deviation of one-year age-sex cohorts	2
OBESE	.103	.305	Dummy variable that equals one if the physician rates the youth as obese or very obese	2
PVIS	.042	.201	Dummy variable that equals one if youth wears glasses and his corrected binocular distance vision is 20/40 or worse or if youth does not wear glasses and his uncorrected binocular distance vision is 20/40 or worse	2
ANEMIA	.023	.149	Dummy variable that equals one if youth is a female whose hematocrit level is more than two standard deviations below the mean for females 12 to 17 years of age or if youth is a male whose hematocrit level is more than two standard deviations below the mean for his stage of sexual maturity	2
B. Preventive Medical Care Measures				
DTPREV	.697	.460	Dummy variable that equals one if youth saw a dentist for a check-up within the past year	1

TABLE 1 (continued)

Variable Name	Sample Mean[a]	Sample Standard Deviation	Definition	Source[b]
DRPREV	.588	.492	Dummy variable that equals one if youth saw a doctor for a check-up within the past year	1
FLDOR	.584	.493	Dummy variable that equals one if the community in which the youth lives uses naturally fluoridated or controlled fluoridated water	See text
C. Other Variables				
FINC	9.614	5.112	Continuous family income (in thousands of dollars) computed by assigning midpoints to the following closed income intervals, $250 to the lowest interval, and $20,000 to the highest interval. The closed income classes are: $500 - $999 $1,000 - $1,999 $2,000 - $2,999 $3,000 - $3,999 $4,000 - $4,999 $5,000 - $6,999 $7,000 - $9,999 $10,000 - $14,999	1
FEDUCAT[d]	11.327	3.227	Years of formal schooling completed by father	1
MEDUCAT	11.142	2.843	Years of formal schooling completed by mother	1
NOPATH	.099	.297	Dummy variable that equals one if youth lives with mother only	1
FLANG	.139	.346	Dummy variable that equals one if a foreign language is spoken in the home	1
LESS20	3.360	1.853	Number of persons in the household 20 years of age or less	1

TABLE 1 (continued)

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
MWORKFT MWORKPT	.268 .154	.443 .361	Dummy variable that equals one if the mother works full-time or part-time, respectively; omitted class is mother does not work	1
DENT	.584	.216	Number of dentists per thousand population in community of residence of youth	See text
PED	.051	.027	Number of pediatricians per thousand population in community of residence of youth	See text
NEAST MWEST SOUTH	.253 .291 .203	.435 .454 .402	Dummy variables that equal one if youth lives in Northeast, Midwest, or South, respectively; omitted class is residence in West	1
URB1 URB2 URB3 NURB	.193 .132 .194 .146	.395 .339 .396 .353	Dummy variables that equal one if youth lives in an urban area with a population of 3 million or more (URB1); in an urban area with a population between 1 million and 3 million (URB2); in an urban area with a population less than 1 million (URB3); or in a non-rural and non-urbanized area (NURB); omitted class is residence in a rural area	1
LMAG	.077	.267	Dummy variable that equals one if the mother was less than 20 years-old at birth of youth	1

TABLE 1 (continued)

Variable Name	Sample Mean[a]	Sample Standard Deviation	Definition	Source[b]
MAG	.096	.294	Dummy variable that equals one if mother was more than 35 years-old at birth of youth	1
LIGHT1	.010	.098	Dummy variable that equals one if youth's birth weight was under 2,000 grams (under 4.4 pounds)	3
LIGHT2	.032	.177	Dummy variable that equals one if youth's birth weight was equal to or greater than 2,000 grams but under 2,500 grams (under 5.5 pounds)	3
BWUK	.245	.430	Dummy variable that equals one if youth's birth weight is unknown	3
FYPH	.117	.321	Dummy variable that equals one if there was a medical difficulty with youth before the age of one year	1
ABN	.200	.400	Dummy variable that equals one if the diagonistic impression of the physician was that the youth had a significant abnormality	2
TWIN	.023	.150	Dummy variable that equals one if youth is a twin	1
FIRST	.497	.500	Dummy variable that equals one if youth is the first born in the family	1
AGE	14.335	1.661	Age of youth	1
MALE	.528	.499	Dummy variable that equals one if youth is a male	1

Footnotes to TABLE 1

[a]The means and standard deviations are computed for the sample of 4,121 white youths described in the text.

[b]The sources are 1 = parents, 2 = examination, 3 = birth certificate. See text for sources of FLUOR, PED and DENT.

[c]The mean of this variable is not zero because standardization was done using the entire Cycle III sample rather than the subsample reported on in this paper. In particular, the negative mean reflects the better oral health of white youths compared to black youths.

[d]For youths who were not currently living with their father, father's education was coded at the mean of the sample for which father's education was reported.

by dentists has a direct impact on tooth decay and periodontal disease. The prescription of eyeglasses by an ophthalmologist or an optometrist can remedy abnormal distance vision. Pediatricians also play an important role in eye care because they often are responsible for examining a youth's eyes initially and referring his parents to an eye specialist if necessary. Finally, all of the health measures excluding vision reflect basic nutritional factors that can be modified by the appropriate diet. These measures are described in detail below.

The periodontal index (APERI) is a good overall indicator of oral health as well as a positive correlate of nutrition (Russell 1956). Kelly and Sanchez (1972, pp. 1-2) describe the periodontal index as follows:

> Every tooth in the mouth ... is scored according to the presence or absence of manifest signs of periodontal disease. When a portion of the free gingiva is inflamed, a score of 1 is recorded. When completely circumscribed by inflammation, teeth are scored 2. Teeth with frank periodontal pockets are scored 6 when their masticatory function is unimpaired and 8 when it is impaired. The arithmetic average of all scores is the individual's [periodontal index], which ranges from a low of 0.0 (no inflammation or periodontal pockets) to a high of 8.0 (all teeth with pockets and impaired function).

It is clear from this description that higher values of the periodontal index correspond to poorer dental health. Our measure, APERI, is scaled somewhat differently from that described above in order to remove the well-known age and sex trends in the periodontal index. APERI is computed as the difference between the adolescent's actual periodontal index and the mean index for his or her age-sex group divided by the standard deviation for that age-sex group.[11] A similar method of age and sex

standardization is used for our other measure of oral health, the number of decayed permanent teeth (IDECAY). We employ two measures of dental health because it is one of the few health problems for which well-defined continuous health measures have been developed.

Obesity is represented by a dichotomous variable that equals one if the physician rates the youth as obese or very obese (OBESE). The physician presumably takes account of the youth's height, age, and sex in making his evaluation.

Anemia is represented by a dichotomous variable that equals one if the youth's hematocrit level is "excessively" low (ANEMIA).[12] The hematocrit level of a female youth is considered to be excessively low if it is more than two standard deviations below the mean for all females 12 to 17 years of age. The hematocrit level of a male youth is considered to be excessively low if it is more than two standard deviations below the mean for all males in his stage of sexual maturity. This procedure is based on Daniel's (1973) findings that (1) hematocrit values differ by sex; (2) these values depend on sexual maturity rather than age for male adolescents; and (3) hematocrit levels are independent of age and sexual maturity for female adolescents.[13]

Abnormal corrected distance vision is denoted by a dichotomous variable that equals one if a youth wears glasses and his corrected binocular distance vision is 20/40 or worse or if a youth does not wear glasses and his uncorrected binocular distance vision is 20/40 or worse (PVIS). This standard of abnormal distance vision is the one used by National Center for Health Statistics (1972).

It is instructive to consider measures of adolescent health that are excluded by our selection criteria. Abnormal hearing is subject to

medical intervention, but the prevalence rate of this condition is less than 1 percent in the HES. Hence, it is far too rare to pose a threat to the future lifetime well-being of a significant percentage of adolescents. High blood pressure is not studied because there is a lack of consensus among pediatricians concerning the importance of this condition in adolescence and the appropriate treatment (National Heart, Lung, and Blood Institute's Task Force 1977). Moreover, the measures of high blood pressure in Cycle III are somewhat suspect (National Center for Health Statistics 1977). Congenital abnormalities are a source of current and future difficulties, but we do not study them because to a large extent they are irreversible. Parental ratings of adolescent health and other subjective indicators are avoided because of the possibility that responses depend on the parents' socioeconomic status. Parents with low levels of income and schooling are likely to be dissatisfied with many aspects of their life including the health of their offspring. Finally, we do not include measures relating to the "new morbidity" such as "learning difficulties and school problems, behavioral disturbances, ... and the problems of adolescents in coping and adjusting ..." [see Haggarty, Roghmann and Pless (1975), p. 316]. While such measures may well reflect life styles that have serious health consequences, they are unlikely to be revealed in a physical exam. Nor are they likely to be easily altered by preventive medical care. Although examination of these and other excluded health measures would be necessary to paint a complete picture of the health of this adolescent cohort, it is not relevant to the objectives of this paper.

B. Measurement of Preventive Dental and Medical Care

Preventive dental care is measured by a dichotomous variable that equals one if the youth saw a dentist for a check-up within the past year (DTPREV). Similarly, preventive pediatric care is measured by a dichotomous variable that equals one if the youth saw a doctor for a check-up within the past year (DRPREV). These variables distinguish between two groups of adolescents: (1) those who received preventive care; and (2) those who received no care at all or only curative care. These two measures of preventive care are preferred to alternatives like the number of dental or physician visits or the receipt of curative care alone because our measures are less likely to reflect reverse causality from poor health to more medical care. Of course, our measures reflect the possibility that adolescents received treatment as well as an examination, but the appropriate treatment of problems revealed by an annual check-up is an integral component of preventive care.

Fluoridation is indicated by a dichotomous variable that is equal to one if the community in which the youth resides uses naturally fluoridated or controlled fluoridated water (FLUOR). Naturally fluroridated communities are serviced by a water supply system that contains a natural fluoride content of 0.7 parts per million or higher. They are identified by the Division of Dental Health of the National Institutes of Health (1969). Controlled fluoridated communities are those that have adjusted the fluoride content of their water supply systems to the optimum level. They are identified by the Division of Dental Health of the National Institutes of Health (1970). For youths who reside in controlled communities, the fluoridation variable equals one only if the

date on which that youth was examined in the HES succeeds the date on which the community adjusted the fluoride content of its water supply system. This insures that youths in controlled communities actually were exposed to fluoridated water.[14]

C. The Pediatrician and Dentist Availability Measures

The youths in Cycle III were selected from 38 distinct primary sampling units. The primary sampling unit is a county or a group of several contiguous counties, some of which form a standard metropolitan statistical area. We obtained data on the number of dentists per capita (DENT) in each youth's primary sampling unit (hereafter termed his county or community of residence) for the year 1968 (the mid-year of the Cycle III survey) from publications of the American Dental Association. The number of pediatricians is not available for the years during which the HES was conducted (1966-70). Therefore, we use the number of pediatricians per capita in the county of residence (PED) for the year 1964 from the American Medical Association (Theodore and Sutter 1965).[15] We believe that the number of pediatricians in 1964 is a good proxy for the number in 1968. Although youths receive medical care from other types of physicians--general practitioners, internists, and ophthalmologists--these physicians also service adults while pediatricians do not. Therefore, we focus on pediatricians as the most important suppliers of physicians' services to youths.[16]

D. Measurement of Other Explanatory Variables

Many of the remaining explanatory variables called for in Section II require no further elaboration. Parents' educational attainment and family

income, for example, are adequately described in Table 1. Some of the other variables listed in Table 1, however, do require additional explanation.

Family size is represented by the number of people in the family who are under 20 years of age at the time of the Cycle III interview (LESS20). Consequently, it may overstate or understate actual completed family size.

Three measures of the family's efficiency in producing healthy children are included in addition to the parents' educational attainment. These are dichotomous variables that identify youths whose mothers were under age 20 when the youths were born (LMAG), youths from homes in which a foreign language is spoken (FLANG), and youths who live with their mothers only (NOFATH). Young mothers are notoriously less efficient at contracepting and may be similarly less efficient in producing healthy offspring. Foreign born families are likely to exhibit differences in household productive efficiency. The absence of her spouse from the household is likely to hinder the mother's allocative efficiency in selecting the input mix with which to produce health. The absence of a father also impinges upon the amount of time that a mother can spend with her children.[17]

The youth's endowed health status is represented by four variables relating to his early health. The first two (LIGHT1, LIGHT2) are dummy variables identifying youths of low birth weight. Low birth weight is a typical indicator of a less healthy birth outcome (for example, Birch and Gussow 1970). Birth weight was obtained from the youth's birth certificate. Since birth certificates are missing for approximately 25 percent of the sample and since we do not focus on the effects of birth weight, we do not delete these observations. Instead, we include a

dummy variable that identifies youths with missing birth certificates (BWUK) in the regression estimates. The third endowment measure is a dummy variable identifying youths whose mothers were over 35 years old at the youth's birth (HMAG). The rationale for including this variable is that older mothers are more likely to have offspring with health defects. The last of these measures is a dummy variable which identifies youths whose parents reported a medical difficulty with the youth before the age of one year (FYPH). Although parents' reports of youths' medical problems before the age of one year are subject to recall error, the first year of a child's life is likely to stand out in his parents' minds relative to other stages in his life cycle. Therefore, we believe that the measurement error in this variable is small.

Our current health indicator is used as a proxy for the child's unmeasured genetic health endowment and his health history beyond age one. This indicator is the presence of at least one significant abnormality as reported by the HES physician who examined the youth (ABN). Abnormalities include heart disease, neurological, muscular, or joint conditions; other major diseases; and otitis media. Except for the last condition, which constitutes a relatively small percentage of all reported abnormalities, these health problems are to a large extent congenital and irreversible.

We also control for several other characteristics of the youth which are not necessarily health related but may cause him to receive better or worse treatment within the family. They are his birth-order (FIRST) and whether or not he is a twin (TWIN). First born youths (or non-twins) will have greater access to individual parental attention because they

arrived in the family first (or they arrived alone). In addition, the youth's age (AGE) and sex (MALE) are included in regressions in which the dependent variable is not adjusted for age and sex (i.e., when the dependent variable is either obesity, abnormal corrected distance vision, preventive dental care, or preventive pediatric care).

Finally, three region variables (NEAST, MWEST, SOUTH) and four sizes of place of residence variables (URB1, URB2, URB3, NURB) are included to control for regional differences that are not otherwise taken into account. We are agnostic about the nature of these differences, but want to avoid the possibility that the health manpower and fluoridation effects are biased by an omission of unmeasured regional characteristics.

III. Empirical Results

In this section we present estimate equations (1) and (3) and compute the total impact of family characteristics and health manpower availability on adolescent health as given in equation (4). Equations (1) and (3) form a recursive system which can be estimated using single equation techniques as long as $E(u_1 u_3) = 0$. We make this assumption here. Although all the dependent variables except the two oral health measures are dichotomous, the method of estimation is ordinary least squares. Preliminary investigation revealed almost no differences between ordinary least squares estimates and dichotomous logit estimates obtained by the method of maximum likelihood. When the dependent variable is dichotomous the fitted equation can be interpreted as a linear probability function in which the regression coefficient of a given independent variable represents the change in the conditional probability

of poor physical health or receipt of preventive care for a one-unit change in the independent variable. The resultant estimates also embody the assumption that several variables that may be considered endogenous (mother's labor force status and family size, for example) are exogenous to adolescent health.[18] Finally, our estimates cannot be unambiguously interpreted as production functions or derived demand equations because insufficient data forced us to use proxy measures for some of the explanatory variables.

Estimates of the dental health production functions and the preventive dental care demand function are discussed in the first part of this section. The physical health production functions and the preventive pediatric care demand function are discussed in the second part. Both discussions are centered on answering the questions posed in the introduction concerning the roles of the family, preventive care, and health manpower availability in determining adolescent health. In examining the results, it is important to remember that the five health measures (APERI, IDECAY, OBESE, PVIS, ANEMIA) are negative correlates of good health, so that negative effects of independent variables in the production functions reflect factors associated with <u>better</u> health outcomes. The two preventive medical care measures (DTPREV, DRPREV), on the other hand, are positive correlates of care; thus positive effects of independent variables in the demand functions reflect factors associated with higher propensities to obtain preventive care.

A. <u>Oral Health</u>

Estimates of the oral health production functions and the preventive care demand function are in Tables 2 and 3, respectively. When the

TABLE 2
Ordinary Least Squares Estimates of Oral Health Production Functions[a]

Independent Variable	APERI		IDECAY		IDECAY (with APERI)	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
FEDUCAT	-.016	-2.95	-.011	-2.10	-.007	-1.41
MEDUCAT	-.030	-4.80	-.028	-4.67	-.021	-3.57
DTPREV	-.255	-8.47	-0.27	-9.42	-.212	-7.48
FLUOR	-.082	-2.96	-.159	-5.96	-.139	-5.39
FINC	-.007	-2.36	-.015	-4.85	-.013	-4.40
LESS20	.036	4.41	.023	2.90	.014	1.86
MWORKFT	.046	1.47	.088	2.91	.077	2.63
MWORKPT	-.007	-0.20	.055	1.54	.057	1.64
NEAST	.038	1.01	.264	7.26	.255	7.24
MWEST	-.072	-1.99	.183	5.24	.200	5.92
SOUTH	-.057	-1.42	.131	3.38	.145	3.85
URB1	.003	0.10	-.071	-1.85	-.072	-1.93
URB2	.027	0.63	-.028	-0.68	-.035	-0.87
URB3	-.088	-2.36	-.153	-4.25	-.132	-3.78
NURB	-.001	-0.00	-.077	-1.96	-.077	-2.02
LMAG	-.009	-0.17	-.032	-0.67	-.030	-0.64
HMAG	.023	0.47	-.048	-1.10	-.053	-1.26
LIGHT1	.187	1.40	-.127	-0.98	-.172	-1.38

TABLE 2 (concluded)

Independent Variable	APERI		IDECAY		IDECAY (with APERI)	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
LIGHT2	.081	1.10	.123	1.73	.103	1.50
BWUK	.014	0.45	.062	2.07	.058	2.02
ABN	.173	5.39	.032	1.04	-.009	-0.32
FYPH	.023	0.57	-.102	-2.64	-.107	-2.87
NOFATH	.016	0.33	.148	3.29	.145	3.31
FLANG	-.051	-1.28	-.172	-4.45	-.159	-4.27
TWIN	-.023	-0.26	.107	1.26	.112	1.37
FIRST	-.013	-0.47	-.024	-0.88	-.021	-0.79
APERI	-	-	-	-	.240	16.47
CONSTANT	.566		.532		.396	
Adj. R^2	.086		.114		.169	
F	15.85[b]		21.37[b]		31.99[b]	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

TABLE 3
Ordinary Least Squares Estimate of Preventive Dental Care Demand Function[a]

Independent Variable	Regression Coefficient	t-Ratio	Independent Variable	Regression Coefficient	t-Ratio
FEDUCAT	.009	3.22	LIGHT2	.070	1.84
MEDUCAT	.023	7.27	BWUK	.005	0.32
FLUOR	.003	0.20	ABN	-.002	-0.14
DENT	.170	4.05	FYPH	.018	0.88
FINC	.011	6.99	NOFATH	-.025	-1.04
LESS20	-.033	-7.95	FLANG	-.037	-1.79
MWORKFT	-.034	-2.07	TWIN	-.002	-0.00
MWORKPT	.044	2.30	FIRST	.007	0.49
NEAST	.046	2.24	AGE	-.006	-1.53
MWEST	.041	2.17	MALE	-.022	-1.67
SOUTH	-.017	-0.83	CONSTANT	.331	
URB1	-.008	-0.37	Adj. R^2	.150	
URB2	-.016	-0.64	F^b	27.02	
URB3	.003	0.14			
NURB	-.029	-1.37			
LMAG	-.065	-2.53			
HMAG	-.018	-0.75			
LIGHT1	.054	0.79			

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

number of decayed permanent teeth (IDECAY) is the dependent variable, two production functions are estimated. The first contains the same set of independent variables as the periodontal index (APERI) regression, while the second includes APERI as an additional independent variable. It has been suggested by Russell (1956) that variations in APERI result largely from genetic factors. If these genetic factors are correlated with the home environment and imperfectly measured by the health endowment variables, the second regression will give a more accurate estimate of the effects of the home environment on IDECAY than the first. Of course, APERI has an environmental component as well as a genetic component (as is evident from our estimate in Table 2). Therefore, the two IDECAY regressions contain upper and lower bound estimates of the impact of the environment on IDECAY.[19]

Most notable among the results are the large significant impacts of a preventive dental visit on both the periodontal index and the decay index.[20] The coefficient estimates imply that adolescents who did not have a preventive check-up within the past year have periodontal indices and decay scores that are each about .3 of a standard deviation worse than adolescents who received a check-up. When APERI is included in the decay equation, the decay differential between the two groups of adolescents declines to .2 but remains statistically significant. To gauge the magnitudes of these effects, recall that APERI and IDECAY have means of approximately zero and standard deviations of approximately one. Therefore, the oral health differentials associated with absence of preventive care are relatively large; they range from 20 to 30 percent of the standard deviations in the scores. Moreover, the differentials apply to a

substantial proportion of the sample: 30 percent of the youths in the HES did not have a check-up in the past year. These findings underscore the efficacy of preventive dental care.

The results pertaining to a publicly provided form of preventive care--water fluoridation--are also strong. Youths exposed to fluoridated water (FLUOR) have significantly better oral health than other youths at all conventional levels of confidence.[21] The fluoridation differentials are smaller, however, than the corresponding preventive dental care differentials in oral health. For example, the fluoridation coefficient in the periodontal index equation is one-third as large as the preventive dental care coefficient. In the decay equations, the ratio of the two coefficients ranges from three-fifths to two-thirds. Nevertheless, given that the per-child cost of fluoridation is substantially below the cost of a preventive dental visit, this remains a cost-effective method of improving dental health.[22]

Let us turn now to the role of the family in determining adolescent dental health levels. The four characteristics of the family environment we focus on are parents education (MEDUCAT, FEDUCAT), family income (FINC), family size (LESS20), and mother's labor force status (MWORKFT, MWORKPT). An overview of the production function estimates in Table 2 reveals that all six variables have statistically significant effects in the expected directions (with the exception of mother's labor force status in the periodontal index equation). Children of more educated parents have better oral health, as do children from families with higher income; while children whose mothers' are employed full-time or who come from larger families have poorer health. The impacts of these

variables on IDECAY are reduced in absolute value when APERI is held constant, but the pattern of statistical significance is not dramatically altered (only the coefficient of father's schooling becomes insignificant). It is clear, then, that these family characteristics have an important impact on adolescent dental health.

We interpret these findings as evidence that the home environment plays an important role in determining children's health. It can be argued, however, that our results do not really constitute strong evidence in favor of "nurture" because of the likelihood of positive correlations first between these family characteristics and the parents' health and second between the genetically determined components of parents' and childrens' health. Put differently, this argument states that family characteristics such as income or parental education largely reflect genetic health factors. For example, parents who are themselves healthy are more likely to be in the labor force and will have higher earnings. Or, parents who have had a healthy childhood and adolescence are more likely to have attained a higher level of education. Two of our findings, however, cast doubt on the applicability of this argument in our case. First, when we include APERI in the decay equation in an effort to more fully control for genetic factors, we still find that these family environment variables have significant impacts on IDECAY. This is noteworthy because the inclusion of APERI is likely to bias the coefficients of the family environment variables toward zero (see note 19).

A second and stronger reason revolves around the coefficients of the educational attainment of the two parents. If the education effect is primarily genetic, we would expect the coefficients of both mother's and father's education to be equal because both parents make an equal genetic contribution to the child. On the other hand, if the education

effect is primarily environmental, we would expect the impact of the mother's education to be larger because she is the family member most concerned with the children's health care. In Table 2 we observe that in every case the coefficient of mother's education exceeds that of father's education. In addition, despite a high correlation between the two education variables (r = .61), the difference in coefficients is always statistically significant at the 10 percent level.[23] Thus, our results clearly indicate that the family environment, and in particular, the mother's education, plays an important role in producing healthy children.

Besides having an important impact on the production of health, family characteristics work to improve adolescent health by increasing the probability that an adolescent receives preventive care. In Table 3 we see that all six of the family variables have significant impacts on the probability that an adolescent received preventive care. Children from families with higher annual income, more educated parents, and in which the mother works part-time, are more likely to receive preventive care, while children from larger families or families where the mother works full-time are less likely to receive preventive care. As an example of the magnitude of these effects, the probability that a child received preventive care in the previous year increases by about two percentage points for each additional year of education received by the mother and declines by about three percentage points for each additional child in the family. Once again we believe that these results reflect environmental rather than genetic influences: the mother's education coefficient is more than twice as large as the father's

education coefficient and the difference between them is statistically significant (t = 2.82).

To determine the total effect of family characteristics on health--both the direct effect embodied in the production function estimates and the indirect effect that operates through the family's proclivity to obtain preventive care--we compute the total impact of these family characteristics in Table 4. The reported coefficients are analogous to the sum $(\beta_1 + \beta_3 \alpha_1)$ in equation (4).[24] Comparison of the coefficients in Tables 3 and 4 indicates that the total impact is from 10 to 100 percent greater than the "direct" effect alone. We also observe, as before, a large and statistically significant (at the 5 percent level) difference between the impacts of fathers' and mother's education, again lending support to our conclusion that "nurture" matters.[25]

With regard to the role of health manpower, we see that it has a large significant effect on the family's propensity to obtain preventive care for its children (Table 3). An increase of one dentist per thousand population increases the probability that adolescents visited the dentist for preventive care in the previous year by 17 percentage points. This estimate is identical to one obtained by Manning and Phelps (1978) and is insensitive to the exclusion of region and size of place of residence from the equation.[26] The implied effect on adolescent health (assuming that dentist availability has no direct impact on adolescent health but operates only by increasing the family's propensity to obtain preventive care) is given in Table 4 and ranges from -.036 to -.047 of a standard deviation in the dental health measures.[27] Thus, an increase in the number of dentists in an area by one per thousand population is

TABLE 4

Total Impacts (Direct and Indirect) of Selected Variables on Oral Health

Variable \ Oral Health Measure	APERI	IDECAY	IDECAY (with APERI)
FEDUCAT	-.018	-.013	-.009
MEDUCAT	-.036	-.034	-.026
DENT	-.043	-.047	-.036
FINC	-.010	-.018	-.015
LESS20	.044	.032	.021
MWORKFT	.055	.097	.084
MWORKPT	-.018	.043	.048

equivalent in its effect on dental health to an increase in the level of the mother's education by one and one-third years.

It should be noted that the positive impact of dentists on the propensity to obtain a check-up is unlikely to reflect demand manipulation by dentists. The concept of demand manipulation refers to the ability of health personnel to shift the demand curve for their services, when all direct and indirect costs of these services are held constant. In his extensive treatment of this phenomenon, Pauly (forthcoming) shows that the demand manipulation effect should be larger in a sample of consumers with positive utilization than in a sample of all consumers. Moreover, his model gives no basis for expecting a demand manipulation effect in an equation that explains the probability of a check-up. Based on these considerations, we view the dental manpower variable as reflecting the importance of information, entry, travel, waiting, and direct costs in the parents' decision to obtain preventive dental care for their offspring.

Most of the other results in Table 3 are consistent with our expectations and will not be discussed.[28] We do wish to point out, however, that although fluoridation does have a significant impact on dental health, it is not significantly related to the probability of obtaining preventive dental care. This is not surprising since from a theoretical point of view either a positive or negative relationship could be predicted. If fluoridation is regarded as an increase in the child's health endowment, the quantity of care demanded should fall. On the other hand, if the increased endowment also increases the marginal product of preventive care, or if it lowers the psychic costs

of obtaining care by reducing the severity of the tooth decay uncovered by a preventive check-up, a positive effect on the quantity of care demanded would be predicted.[29] Both types of results have been reported in other studies. Manning and Phelps (1978) report mixed effects of duration of exposure to fluoridation on the propensity to obtain preventive dental check-ups for white children below the age of 15 in a 1970 health survey conducted by the National Opinion Research Center. Upton and Silverman (1972) use 1966 data for 15 midwestern towns, half of which used fluoridated water, and report fewer restorations of children's permanent teeth in the fluoridated towns.

We conclude this subsection by using our results to estimate the impacts of three government programs to improve the oral health of youths. First, consider a $1,000 income transfer to low-income families. As shown by the reduced-form coefficients of FINC in Table 4, the transfer would lower the periodontal index of youths from these families by .01 points and would lower their decay index by .02 points. (Such a program would naturally also have other beneficial effects on children and their families.) Next consider a program to reduce or eliminate regional differences in the number of dentists per thousand population. Dentists are more numerous in urban areas than in rural areas. To take two sites in the HES, there were 1.1 dentists per thousand population in San Francisco, California, while there were .2 dentists per thousand population in San Benito, Texas. Suppose that this difference were eliminated by raising the number of dentists in San Benito by one per thousand population. Then the periodontal index of youths in San Benito would fall by .04 points, and their decay index would fall by .05 points.[30] Finally,

consider an 80 percent reduction in the price of a dental check-up as a result of the enactment of a national health insurance plan for dental care with a 20 percent co-insurance rate. Based on research by Manning and Phelps on the impact of price on the propensity to obtain preventive dental care for children and youths, we estimate that such a policy would raise the probability of obtaining care by 16 percentage points. This would improve both the periodontal and the decay scores by .04 points.[31]

We view the above computations as illustrative rather than definitive. To choose among the three programs, information on the cost of each program and on the number of youths affected clearly is required. Moreover, as indicated in Section I, definitive computations of impact effects should take account of the supply elasticity of dental care and the exact nature of the relationship between dental manpower and the indirect costs of obtaining dental care.

B. Physical Health

Estimates of the physical health production functions and the preventive care demand function appear in Tables 5 and 6, respectively. Looking first at the production function estimates, we are struck by the fact that these physical health measures are much less amenable to statistical explanation than are the dental health measures. Of course, lower R^2's would be expected for the three physical health measures because they are dichotomous rather than continuous. But many fewer explanatory variables are statistically significant in the physical health case. Clearly unmeasured genetic or "luck" factors play a much larger role in the case of these health measures.

TABLE 5
Ordinary Least Squares Estimates of Physical Health Production Functions[a]

Independent Variable	OBESE		PVIS		ANEMIA	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
FEDUCAT	.001	0.46	-.002	-1.51	-.001	-0.65
MEDUCAT	-.005	-2.01	.001	0.71	-.002	-2.07
DRPREV	-.005	-0.50	-.005	-0.75	.002	0.47
FINC	.0002	0.20	-.001	-1.04	-.001	-1.39
LESS20	-.007	-2.41	.006	3.31	.004	2.60
MWORKFT	.005	0.40	.001	0.10	.002	0.37
MWORKPT	-.007	-0.54	-.00003	-0.00	.0003	0.00
NEAST	.040	2.94	-.003	-0.36	-.013	-1.93
MWEST	.036	2.80	.012	1.45	-.010	-1.63
SOUTH	-.020	-1.36	.009	0.93	.002	0.24
URB1	-.017	-1.15	-.0002	-0.00	.007	0.96
URB2	.023	1.47	.010	0.91	.010	1.33
URB3	-.011	-0.79	-.009	-1.01	.015	2.27
NURB	.016	1.09	-.008	-0.85	.014	1.94
LMAG	.023	1.27	-.011	-0.92	.005	0.54
HMAG	.017	1.01	.002	0.20	.003	0.40
LIGHT1	-.001	-0.00	.003	0.10	-.020	-0.84

TABLE 5 (concluded)

Independent Variable	OBESE		PVIS		ANEMIA	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
LIGHT2	-.010	-0.36	-.003	-0.17	-.010	-0.73
BWUK	.0004	0.00	-.012	-1.59	-.013	-2.39
ABN	.122	10.38	.009	1.17	-.008	-1.40
FYPH	-.006	-0.40	.006	0.59	.001	0.10
NOFATH	.021	1.22	.009	0.81	.006	0.71
FLANG	.015	1.06	.021	2.13	.001	0.20
TWIN	.004	0.10	.017	0.79	-.020	-1.25
FIRST	.021	1.95	.008	1.20	.003	0.66
AGE	-.004	-1.23	-.008	-4.28	-	-
MALE	-.053	-5.65	-.024	-3.87	-	-
CONSTANT	.190		.164		.049	
Adj. R^2	.039		.013		.008	
F	7.21[b]		2.97[b]		2.35[b]	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

TABLE 6
Ordinary Least Squares Estimate of Preventive Pediatric Care Demand Function[a]

Independent Variable	Regression Coefficient	t-Ratio	Independent Variable	Regression Coefficient	t-Ratio
PEDUCAT	.013	4.28	LIGHT1	-.031	-0.40
MEDUCAT	.007	1.82	LIGHT2	.063	1.47
PED	.675	1.97	BWUK	.001	0.10
FINC	.005	2.59	ABN	.094	5.00
LESS20	-.017	-3.60	FYPH	.042	1.81
MWORKFT	.015	0.80	NOFATH	-.071	-2.61
MWORKPT	.005	0.24	FLANG	.017	0.72
NEAST	.062	2.77	TWIN	-.025	-0.49
MWEST	-.031	-1.50	FIRST	.058	3.47
SOUTH	-.021	-0.91	AGE	.004	0.95
URB1	.021	0.87	MALE	.064	4.30
URB2	.036	1.34	CONSTANT	.192	
URB3	-.014	-0.61			
NURB	-.037	-1.56	Adj. R^2	.067	
LMAG	-.032	-1.09	F	11.99[b]	
HMAG	-.055	-2.09			

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

In contrast to the results for preventive dental care, there is little evidence that preventive medical care is efficacious. Youths who saw a doctor for a check-up within the past year (DRPREV) have one-half percentage point smaller probabilities of being obese or of having abnormal corrected distance vision than other youths, and a one-fifth percentage point higher probability of having anemia. None of these three differentials is statistically significant. One possible explanation for these findings is that there are fairly long lags between the receipt of preventive care and an improvement in physical health. Alternatively, one might argue that physicians play a minor role in the outcomes studied here relative to unmeasured endogenous inputs such as proper diet. The non-significant impact of preventive care also means that family characteristics operate on health only through the production function. There are no indirect effects of the various family characteristics on physical health, only direct effects. Consequently, we do not present a table of "total" effects (comparable to Table 4) in the case of the physical health measures.

The relationship between family characteristics and health is also much weaker in the case of physical health. Most of the six family characteristics variables studied are not even statistically significant in the production function; only the mother's education and family size variables have significant impacts. Children of more educated mothers are less likely to be obese or anemic, and they are more likely to have poor vision (the latter relationship is not significant). Children from larger families are more likely to have poor corrected vision or be anemic, but they are less likely to be

obese. To get an impression of whether these effects can be viewed as environmental as opposed to genetic, we again look at the difference between the coefficients of the two parent's education variables. For both OBESE and ANEMIA, the mother's education coefficient is larger than the corresponding father's education coefficient, but for PVIS the opposite is true. Only in the case of obesity is the difference significant at the 10 percent level. Thus, in this case the evidence regarding a nature versus nurture interpretation of the family effects is not conclusive, but it does suggest that with respect to obesity at least one component of the family environment--mother's education--has an important impact.

We noted that the family size variable has a perverse sign in the obesity equation: children from larger families are in better rather than worse health in that they are less likely to be obese. The positive relationship between family size and the incidence of the other health problems is easy to rationalize (it may reflect a substitution away from higher "quality" children as the shadow price of quality rises),[32] but a justification for the negative relation reported for obesity is less obvious. One possible explanation for this negative family size effect (as well as for the positive income effect) is the existence of joint production among various aspects of quality. For example, families with fewer children or higher income may consume more rich and caloric foods. This consumption raises some aspects of quality but at the same time makes obesity more likely.

The finding of non-significant effects of family income in physical health outcomes has important implications. First, it suggests that

policies to improve the well-being of adolescents via income transfers would have little impact on our physical health measures. Second, this finding coupled with the significance of the mother's schooling variable underscores the key role in health production of nonmarket productivity as opposed to market goods and services as measured by family income and preventive care. This result echoes our earlier findings for a group of younger children (see Chapter 2). In the case of obesity, we believe that the impact of mother's schooling reflects the information that highly educated mothers have acquired as part of the schooling process about the dangers of obesity and about what constitutes an appropriate diet.

Family effects in the derived demand for preventive pediatric care (Table 6) tend to be much stronger than they are in the production functions (although the R^2 in the preventive pediatric care equation is still substantially lower than in the preventive dental care equation). Among the six family variables, only the mother's labor force status variables do not have a significant impact on the family's probability of obtaining preventive care. Families with higher parental education and more income are more likely to get preventive care for their children while larger families are less likely to. In addition, father's education has a larger impact than mother's education. It is not clear how to interpret these results, however, since we have no evidence that preventive pediatric care is efficacious.

The last result to be discussed concerns the role of pediatrician availability. Similar to the corresponding findings for preventive dental care, the number of pediatricians per thousand population in the

county of residence (PED) has a positive and statistically significant regression coefficient in the demand curve for pediatric care. This finding complements those reported by Kleinman and Wilson (1977) and Colle and Grossman (1978). However, the implied effects of an increase of one pediatrician per thousand population are small (-.003, -.003, and .001 for OBESE, PVIS, and ANEMIA, respectively), primarily because the health impact of preventive care is small and not significant. Thus our findings indicate that a policy to increase pediatric manpower in medically underserved areas would not improve the physical health of adolescents—at least as represented by our three measures. Such a policy should be given a much lower priority than a analogous policy to expand dental manpower in areas characterized by shortages.[33]

IV. Summary and Implications

The purpose of this study has been to examine the determinants of the oral and physical health of white adolescents with special emphasis on the roles of family background and the use of preventive medical care. The main results of the study are (1) nurture plays an important role in determining oral health but less so for the other health problems studied; (2) preventive care is efficacious in the case of oral health but not for the other health problems studied; and (3) the three physical health measures are largely unexplained by the family and preventive care variables used here. Only mother's education and family size have significant impacts.

With respect to the first result, mother's schooling is singled out as a crucial component of the home environment. Although mother's schooling, father's schooling, family income, and family size all make

significant contributions to oral health, mother's schooling dominates father's schooling. Moreover, mother's schooling tends to dominate both income and father's schooling in the physical health equations, especially in the case of obesity. The finding that the impact of mother's schooling almost always exceeds that of father's schooling is especially important because equal effects would be expected if the schooling variables were simply proxies for unmeasured genetic endowments.

Two additional pieces of evidence underline the robustness of the finding that nurture "matters." First, the relative magnitude of the effect of the various family background variables on the index of tooth decay is not greatly altered when the periodontal index, a proxy for genetic oral health endowment, is held constant. Second, the identification of a plausible mechanism by which family characteristics influence adolescent health--preventive care--increases our confidence that these variables reflect a behavioral effect as opposed to a genetic effect or a statistical artifact.

With regard to the role of preventive dental care, youths who received a preventive dental check-up within the past year and youths exposed to fluoridated water have much better oral health than other youths. Moreover, the probability of a preventive examination is positively related to the number of dentists per capita in a youth's county of residence. This implies that a program to increase the availability of dentists in medically deprived areas would improve the oral health of youths in these areas. Indeed, we estimate that the payoffs to increasing dental manpower by one per thousand population are about the same as the payoffs to the coverage of preventive dental care under national health insurance.

The probability of obtaining a preventive check-up by a doctor is also positively related to family income and to the number of pediatricians per capita in the county of residence. But we have little evidence that preventive care delivered to youths by physicians is efficacious in terms of their physical health. Therefore, the payoffs to national health insurance for physicians' services delivered to youths or programs to increase the availability of doctors who treat youths are very small.

Our results for the physical health measures are weak, but one pair of findings does stand out. Adolescents are less likely to be obese if their mothers are highly educated, and they are more likely to be obese if they come from small families. The latter relation provides a partial explanation of the dramatic increase in obesity during recent decades since over the same period we have seen a startling decline in family size. The former relation, on the other hand, suggests a strategy for slowing down the trend in the incidence of this health problem. What is needed is a public information program--similar to that mounted in the case of childhood immunization--directed at alerting less educated parents, and especially mothers, to the dangers associated with childhood obesity.

Overall, what our results suggest is that selective rather than general programs would be most effective in improving the health of the population under 18 years of age. For instance, instead of providing complete coverage for physicians services delivered to persons from birth to age 18 under national health insurance, the government should direct its attention at prenatal care and physicians services

during the first year of life. It is known that appropriate prenatal and infant care can make a difference in terms of health outcomes (for example, Lewit 1977). Conversely, our results for oral health in this study and in our previous research (see Chapter 2) suggest that the payoffs to the coverage of dental care from the age it is first received until age 18 or beyond would be substantial.

FOOTNOTES

[1]For a partial survey of this literature, see Grossman (1975) and Edwards and Grossman (1979).

[2]A full description of the sample, the sampling technique, and the data collection is presented in National Center for Health Statistics (1969).

[3]Children's health also depends on the prices of inputs used to produce other aspects of their quality and the prices of other forms of parents' consumption. The effects of these variables will not be studied here.

[4]For discussions of the indirect costs of obtaining pediatric care, see Colle and Grossman (1978) and Goldman and Grossman (1978).

[5]One possible objection to using this type of framework to analyze the health of adolescents is that the goals of parents and youths are likely to differ. For instance, cigarette smoking by a youth might increase his utility but reduce his parents' utility because it is detrimental to his current or future health. This type of conflict between parents and youths has been analyzed by Becker (1974) in the context of an economic model of social and family interactions. He shows that such conflicts are important when the parents' utility function depends on particular "merit" commodities consumed by the youth rather than on his consumption of all commodities. In such a case parents have an incentive to allocate resources not only to their children's consumption, but also to policing their offsprings' consumption patterns. An explicit melding of our model with Becker's would be a difficult task, and although it would alter the interpretations of the effects of various family characteristics, it would not add to or delete from the list of relevant explanatory variables.

[6]Given the essentially arbitrary scaling of all of our adolescent health measures and the general ignorance concerning the exact specification of a health production function, we believe that it is inappropriate to experiment with more sophisticated functional forms.

[7]The Bureau of Labor Statistics does collect measures of the prices of various goods and services, including physician and dental office visits, for 40 cities and four nonmetropolitan areas. We do not take price variables from this source because they are based on small samples.

and the sites in the HES survey are not identical to the sites in the BLS survey. On the other hand, the number of dentists and pediatricians are based on complete enumerations in all counties by the American Dental Association and the American Medical Association and can be matched easily to the HES sites. Thus the two manpower variables have little measurement error, while the price estimates from the BLS would contain a great deal of measurement error.

[8]This is in contrast to the work of Newhouse and Friedlander (1977) who fit an equation similar to our equation (4).

[9]The term "allocative effect" and the decomposition of the schooling parameter into direct and allocative components is due to Welch (1970). He uses this framework to study the impact of schooling on market production. Technically, schooling is a relevant determinant of the demand for medical care even if it has no allocative efficiency effect. In simple models of schooling as an efficiency variable in household production (Grossman 1972; Michael 1972), schooling raises the amount of health output obtained from a given vector of inputs. In such models schooling can lower the quantity of medical care demanded at the same time as it raises the quantity of health demanded. In particular, medical care would rise only if the income and price elasticities of demand for offspring's health exceeded unity. We stress a model that incorporates an allocative efficiency effect because schooling should increase the parents' knowledge about what constitutes an appropriate diet, when to take their children to the doctor or the dentist for a preventive check-up, how to follow the doctor's advice, and how to

foster appropriate oral hygiene behavior by their children. The ability of parents with extra schooling to select a better input mix, as well as to obtain a larger health output from given inputs, is likely to encourage them to demand larger quantities of preventive care even if the income and price elasticities of health are less than one. In part the effect may reflect a substitution toward preventive care and away from curative care.

[10]In adopting these two criteria for the selection of health measures, we are guided in part by Kessner's (1974) tracer methodology for studying the health of children and adolescents.

[11]If the actual periodontal index of each age-sex group is normally distributed, APERI could be translated directly into the youth's periodontal index percentile. We have experimented with the actual value of the periodontal index as the dependent variable in a multiple regression that includes age, the square of age, and a dummy variable for male adolescents in addition to the remaining independent variables. The results obtained (not shown) are similar to those reported in Section III.

[12]Dutton (1978) advocates the use of a continuous, rather than a discrete, measure of anemia. She conducts a multiple regression analysis of actual hematocrit levels of black children between the ages of 6 months and 4 years. The only statistically significant variables in this regression (at the 5 percent level) are age and sex. Therefore, it is not at all clear what we would gain by adopting her measure.

[13]Similar patterns are present in the Cycle III data. Tanner (1962) stresses the importance of sexual maturity in the determination of the health and cognitive development of adolescents. Preliminary analysis revealed, however, that sexual maturity does not have an effect on our health measures except in the case of hematocrit levels of females.

[14]Clinical evidence suggests that exposure to fluoridated water is particularly important if it occurs during the ages at which the permanent teeth are being formed (McClure 1962). These teeth do not appear until a child is approximately 6 years-old but start to be formed a few months after birth. Therefore, it is useful to identify youths who had been exposed to fluoridated water before they reached the age of 6 years. Unfortunately, we cannot do this because the youth's current residence alone is reported in the HES. We did create a fluoridation variable that identifies youths exposed before age 6 under the assumption of no migration, but it had no effect on oral health in regressions that included the fluoridation variable described in the text.

[15]Our measure of the number of dentists excludes those in the Federal dental service. The number of pediatricians pertains to those in private practice.

[16]Since pediatricians treat only children and youths, the number of pediatricians per person under a certain age (say age 18) might appear to be a more relevant measure than the number of pediatricians per capita. We did not employ such a variable for several reasons. First, the appropriate age cutoff is not obvious. Second, even if pediatricians do not treat youths beyond the age of 17, since mothers

typically are responsible for taking youths to the physician, the indirect costs of obtaining pediatric care might be more related to the number of pediatricians per woman with children below the age of 18 than to the number of pediatricians per person below the age of 18. Third and most important, there is little variation in persons under age 18 as a percentage of the population or in women with children under age 18 as a percentage of the population among the 38 sites in the HES.

[17]The educational attainment of absent fathers is not known. For children with absent fathers, we code FEDUCAT at the mean level of father's education in the subsample of youths who live with both parents. This coding scheme is consistent with the assumption that father's education has the same relationship with adolescent health whether or not the father is actually present. An alternative assumption is that father's education has no affect on adolescent health if he is absent. Under this assumption, the education of absent fathers would be coded at zero. Use of the alternative coding scheme would alter the regression coefficient of NOFATH but would not alter the coefficient of FEDUCAT or the coefficients of other independent variables in the regression.

[18]The health endowment variables are also endogenously determined because they are affected by family choices regarding prenatal care, timing of childbearing, and resources allocated to children since birth. Despite the endogeneity of the health endowment measures, mother's labor force status, and family size, preliminary computations revealed that the estimated coefficients of the other family background measures and

of preventive care are only slightly altered by the exclusion of these variables from the equations.

[19]Suppose that the periodontal and decay functions are

$$\text{APERI} = a_1 G + a_2 E + u_1 \qquad (1)$$

$$\text{IDECAY} = b_1 G + b_2 E + u_2 \quad , \qquad (2)$$

where G is genetic oral health endowment, E is the home environment, u_1 and u_2 are disturbance terms, and intercepts and other independent variables are ignored. Note that a_1, a_2, and b_2 are negative since a more favorable endowment or environment improves oral health. Solve equation (1) for G and substitute into equation (2) to obtain

$$\text{IDECAY} = b_1 a_1^{-1} \text{APERI} + (b_2 - a_2 b_1 a_1^{-1}) E + u_2 - b_1 a_1^{-1} u_1 . \qquad (3)$$

Clearly, the absolute value of the parameter of E in equation (3) is smaller than the absolute value of the corresponding parameter in equation (2). Note that APERI is negatively correlated with the composite disturbance term $(u_2 - b_1 a_1^{-1} u_1)$ in equation (3). Therefore, if the equation is estimated by ordinary least squares, the regression coefficient of APERI is biased toward zero and that of E away from zero provided E and APERI are negatively related. In the text we make the plausible assumption that this upward bias in the absolute value of the regression coefficient of E is offset by the fundamental difference between the structural parameters of E in equations (2) and (3). That is

we assume that the expected value of the regression coefficient of E understates $|b_2|$ even though it overstates $|b_2 - a_2 b_1 a_1^{-1}|$.

[20]Statements concerning statistical significance in the text are based on one-tailed tests except when the direction of the effect is unclear on a priori grounds or when the estimated effect has the "wrong sign." In the latter cases two-tailed tests are used.

[21]The estimated effects of fluoridation on oral health are not sensitive to the omission of the three region and four size of place of residence variables from the regressions. This indicates that the fluoridation variable is not simply a proxy for location.

[22]Consumer Reports (1978) cites a report in the New England Journal of Medicine which estimates the per capita cost of fluoridation to be about 10 to 40 cents per year (p. 393).

[23]The relevant "t" statistics for the three equations in Table 2 are 1.41, 1.79, and 1.48. Note that probable biases in the estimates of the two parents' education coefficients are likely to work towards a finding of no significant difference. The estimate of the direct efficiency effect of father's schooling may be biased away from zero; and the estimate of the direct efficiency effect of mother's schooling may be biased toward zero. The former bias is introduced if father's education serves as a proxy for permanent income (if there is measurement error in current family income). The latter bias is introduced if more educated mothers allocate less time to the production of adolescent oral health because they have a higher opportunity cost of time, and if

the opportunity cost of time effect is not fully reflected by the two measures of mother's labor force status. Along similar lines, the estimated father's education effect may be biased upward in the demand curve for preventive care. The mother's education effect is biased downward if oral health is "time-intensive" and if substitution in consumption outweighs substitution in production.

[24]These could be thought of as solved "reduced form" coefficients of the exogenous variables.

[25]As is expected on the basis of the education coefficients in Tables 2 and 3, the difference in "total" effects is larger than the difference in direct effects. The test of the significance of the difference between the "total" effect of mother's schooling and the "total" effect of father's schooling is based on the estimated reduced form--the ordinary least squares regression APERI or IDECAY on all the exogenous variables. This procedure is employed because standard errors of solved reduced-form coefficients and standard errors of difference between such coefficients are very difficult to compute. In every case, the estimated reduced-form difference between the schooling coefficient is exactly the same as the solved reduced-form difference. Therefore, the bias introduced by our test is minimal. The test statistics are 1.81, 2.23, and 2.82 for APERI, IDECAY, and IDECAY with APERI, respectively.

[26]Manning and Phelps estimate a discriminant function of the probability of obtaining a check-up. They point out that the coefficients in this equation approximate logit coefficients. Since they do not

indicate the mean probability of a check-up in their sample, we converted their logit coefficient of the number of dentists into a marginal effect at the mean check-up probability in the HES sample of .7. If m is the marginal effect of a given independent variable, b is its logit coefficient, and p is the probability of a check-up, the conversion formula is

$$m = bp(1 - p) \quad .$$

[27]The finding that the periodontal index is inversely related to the number of dentists differs from that of Newhouse and Friedlander (1977). Using adults in Cycle I of the HES, they report an insignificant positive effect of dentists per capita in the county of residence on the periodontal index. Their result is based on an ordinary least squares regression of the periodontal index on the number of dentists and other variables and does not embody the restrictions discussed in Part C of Section I.

[28] There are two "perverse" results that are statistically significant: youths from families in which a foreign language is spoken in the home (FLANG) have better oral health than other youths; and youths whose parents reported a medical difficulty with the youth before the age of one year (FYPH) have less decay than other youths. The first of these may be caused by genetic differences in oral health between native Americans and immigrants or the native-born offspring of immigrants. We offer no explanation for the latter finding.

[29]For a general discussion of endowment effects in models such as the one employed in this paper, see Tomes (1978). A detailed treatment of the role of fluoridation in dental care demand functions appears in Upton and Silverman (1972).

[30]The reduction in the decay score is taken from the reduced-form coefficient of DENT obtained from the decay function that excludes APERI.

[31]In their discriminant estimate of the decision for white children and youths to receive a dental exam, Manning and Phelps specify a price effect that varies with family income. Our extrapolation of their results assume (1) that family income equals $10,000 (the mean value in the HES), (2) that the uninsured price of check-up is $15, and (3) that the uninsured probability of a check-up is .7 (the mean in the HES). The reduction in the decay score is obtained from the decay function that excludes APERI.

[32]Alternatively, these effects may be attributed to a reduction in per capita income as family size rises with family income held constant. Indeed, the sign of the family size effect is opposite that of the family income effect in all three regressions. Yet something more than a mechanical relationship between family size and per capita income is required to account fully for the contribution of family size to health outcomes. For example, unlike the family size coefficients, the family income coefficients are not always statistically significant. In addition, computations reveal that the impact on physical health of a 1 percent

increase in family size is larger in absolute value than that of a 1 percent increase in family income.

[33] Some readers may object to the constraint in our recursive model that the direct effect of health manpower on health is zero. For the benefit of these readers, the estimated reduced-form coefficients of the number of pediatricians on obesity, abnormal vision, and anemia are -.316 (t = -1.47), -.010 (t = -0.07), and -.168 (t = -1.57), respectively The estimated reduced-form coefficients of the number of dentists on the periodontal index and decay are .128 (t = 1.55) and .085 (t = 1.08), respectively. These coefficients give a very different and, in our view, inappropriate picture of the payoff of a program to expand pediatric manpower compared to a program to expand dental manpower.

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Chapter 5

THE DYNAMICS OF HEALTH AND COGNITIVE DEVELOPMENT IN ADOLESCENCE

The importance of family background in the determination of the health and cognitive development of children and adolescents and the existence of cross-sectional relationships between health and cognitive development have been well established in a number of studies. Edwards and Grossman (see Chapter 2) emphasize the key role of parents' schooling in the health outcomes of white children between the ages of 6 and 11 in Cycle II of the U.S. Health Examination Survey (HES). They report similar findings in a study of health outcomes of white adolescents between the ages of 12 and 17 in Cycle III of the HES (see Chapter 4). In particular, they indicate that mother's schooling has a larger impact on adolescent health than father's schooling. Edwards and Grossman (1979) demonstrate a number of strong relationships between children's health and cognitive development in Cycle II of the HES. In addition, they summarize comparable findings for underdeveloped countries, Great Britain, and poverty populations in the United States. Of course, there is a massive literature on the beneficial impacts of a favorable family background on children's cognitive development.[1]

Especially in the case of health, the extent to which family background effects reflect the importance of the home environment (nurture) as opposed to genetic inheritance (nature) remains an open issue.[2] Moreover, cross-sectional regressions of cognitive development on health, such as those estimated by Edwards and Grossman (1979) and others, do

not establish causality from health to cognitive development. Statistically significant relationships may reflect causality that runs from cognitive development to health as well as causality from health to cognitive development. To be concrete, poor health may impede the ability to learn, thereby resulting in lower scores on ability tests; and slow intellectual growth may impinge on a youth's efficiency in the production of his health. Simple cross-sectional correlations do not reveal which, if any, of the causal relations is in operation. Indeed, the correlation may arise not because of any structural causality but because both health and cognitive development are linked to unmeasured genetic factors.

The purposes of this study are (1) to estimate the pure environmental contribution of family background in general and parents' schooling and family income in particular to the health and cognitive development of adolescents and (2) to examine whether health causes cognitive development, cognitive development causes health, or both cause each other. The study utilizes a panel data set that contains measures of the health and cognitive development of adolescents, corresponding measures of their health and cognitive development as children, and family background variables. The sample consists of the adolescents in Cycle III of the Health Examination Survey who were also examined in Cycle II when they were children.

To study the dynamics of health and cognitive development and to deal with the problem of simultaneity, we investigate the causal priorness of these measures. Specifically, we assume that the processes that generate them are Markov and can be estimated by a simple first-order

ARMA model. We show that, if the genetic impact on these variables is restricted to the determination of initial conditions, then the estimates of the time paths will be free of genetics-bias and will reflect the true environmental effects of family background variables. Empirically, we estimate two multivariate equations: one relating adolescent health to childhood health, childhood cognitive development, and family background; and a second relating adolescent cognitive development to childhood cognitive development, childhood health, and family background. In the health equation, the coefficient of family background gives an estimate of the environmental effect that controls for the initial or inherited level of health. The coefficient of childhood cognitive development indicates whether the latter variable causes adolescent health. The adolescent cognitive development equation can be interpreted in a similar manner. In particular, the coefficient of childhood health measures the significance and size of the causal relationship from health to cognitive development.[3]

Clearly, the notion of causality implicit in our research is akin to that of Granger (1969). Specifically, cognitive development causes health if the prediction of adolescent health conditional on childhood health is improved by information on childhood cognitive development. Similarly, health causes cognitive development if the prediction of adolescent cognitive development conditional on childhood cognitive development is improved by information on childhood health.

I. Econometric Model

In related research, Edwards and Grossman (1979, and also Chapters 2 and 4) have argued that offspring's health and cognitive development can be viewed as aspects of their quality and can be examined fruitfully within the context of economic models of fertility and household production developed by Becker (1965), Becker and Lewis (1973), Willis (1973), and Becker and Tomes (1976). These models provide useful theoretical insights with regard to the designation of a set of factors as determinants of health and cognitive development and with regard to the predicted direction of effects. The fundamental problem in analyzing the determinants of health and cognitive development empirically is to separate the genetic effects from those of observed socioeconomic and background variables. We suppose that at any point in time a child's health can be characterized by a random variable whose distribution is conditional on the child's cognitive development, the initial health of the child, family socioeconomic variables, and some genetic endowment, and that a child's cognitive development can be characterized by a random variable whose distribution is conditional on the child's health and the same set of background variables. Many of these variables, of course, are themselves partially determined by the genetic endowments of both the parents and the child. This scenario is recognizable as the multiple-indicator/multiple-cause model, where some of the indicators are themselves causes. But rather than proceeding directly to multi-equation models of this type, we restrict our attention in this study to models which can be analyzed using single-equation methods.

Briefly stated, the usual methodology designed to deal with unobserved components strives to identify some systematic component and extract its effects from the conditional distribution of the dependent variable in question. The identification problem is most serious in a single-equation cross-section specification, since it is generally impossible to distinguish the systematic unobserved component from the random error entering the equation. One approach has been to include indicators of the unobserved variable which are not themselves part of the original cross-section specification, under the presumption that these indicators serve as instruments for the unobserved variable. An example is the inclusion of test scores as a proxy for ability in earnings equations. But investigators generally acknowledge that this is a second-best procedure, since it introduces an errors-in-variables bias which may be nearly as large as the original omitted-variables bias.[4]

More recent attempts to estimate models with unobserved variables have been hybrids of multi-equation models. Identification is achieved by presuming that the unobserved variable is reflected in more than one observation on an indicator variable, either across individuals[5] or in the same individual observed over a number of time periods.[6] Estimation procedures have usually followed either a fixed or random effects approach to deal with the unobserved variable.[7] The specific nature of the health/cognitive development question, however, makes both of these methods impossible or impractical.

The fixed effects approach assumes that each individual's probability distribution is indexed by an unobserved nuisance parameter, as well as some set of observed explanatory variables. In our case, we might

label this nuisance parameter as genetics. If we have more than one observation from each individually-indexed distribution, then this nuisance parameter can be estimated directly together with the other unknown parameters of the distribution function which are common across individuals. However, an identification problem arises if the distribution functions are also indexed by a variable or variables which have the same time structure as that presumed for the unobserved variable to be estimated. In our case, several of the background variables are time-invariant, such as parents' education and birth weight; it is impossible to distinguish an estimate of the affects of an unobserved time-invariant component such as genetics from the effects of these observed variables. Linear models presuming a fixed effect and non-zero effects of time-invariant observed variables will be observationally equivalent to models presuming only a fixed effect.

To illustrate the preceding point, let $\tilde{y}_i$ denote a vector of T observations on some variable y_i for individual i, and suppose that the distribution of $\tilde{y}_i$ is described by the linear model

$$\tilde{y}_i = \tilde{X}_i \theta_1 + \tilde{Z}_i \theta_2 + \tilde{\lambda}_i + \tilde{\epsilon}_i \quad , \tag{1}$$

where X_i is a Txk_1 matrix of time-varying independent variables, Z_i is a Txk_2 matrix of time-invariant independent variables, $\tilde{\lambda}_i$ is the vector of fixed effects with each element being λ_i, and $\tilde{\epsilon}_i$ is a vector of i.i.d. errors. Suppose that λ_i and θ_2 are unknown. Then the distribution given by (1) is observationally equivalent to one given by

$$\tilde{y}_i = \tilde{X}_i \theta_1 + \tilde{Z}_i \theta_2^* + \tilde{\lambda}_i^* + \tilde{\epsilon}_i \quad , \tag{2}$$

where

$$\tilde{\lambda}_i^* = \tilde{\lambda}_i + \tilde{Z}_i (\theta_2 - \theta_2^*) \quad .$$

It is important to add that this inability to identify λ_i and θ_2 simultaneously in (1) does not mean that an unbiased estimate of θ_2 can be obtained by omitting the unknown fixed effect from (1), unless we maintain a hypothesis that λ_i is uncorrelated with both $\tilde{X}_i$ and $\tilde{Z}_i$. This is not a reasonable hypothesis in the case of genetics and background variables.

This identification problem can be avoided by using a random effects model and treating $\tilde{\lambda}_i + \tilde{\varepsilon}_i$ as a composite error term with covariance structure

$$\Sigma = \sigma_\lambda^2 \; i\,i' + \sigma_\varepsilon^2 \; I \quad ,$$

where i is the column vector of units, σ_λ^2 is the population variance of the unobserved systematic component, and σ_ε^2 is the variance of the true error term. If Σ is known, then consistent estimates of θ_1 and θ_2 can be obtained by estimating (1) with GLS. The problem is, however, that Σ is not observed, nor can it be consistently estimated without resorting to a model with fixed effects, which as we have shown above is not estimable.[8]

An alternative to these cross-section formulations, which may circumvent the identification problem in the presence of the unobserved genetic component, is to express health and cognitive development as explicitly dynamic processes and to hypothesize that the timing of genetic

and socioeconomic effects differ. A major shortcoming of cross-section models, whether they use one or more observations per individual, is that they fail to take account of dynamics which arise naturally in most economic problems, either in terms of structural or temporal dependencies for decision making in a dynamic context. Theories of human capital have stressed the life cycle nature of an individual's economic situation, with economically-relevant variables having a time as well as an individual index. If uncertainty is added, this life-cycle characterization is tantamount to depicting an individual's economic life (including measures of health and cognitive development) as a vector-valued stochastic process.

It is worthwhile to consider a simple dynamic formulation to assess its suitability for modeling health and cognitive development. Consider a variable h(t) which represents the number of individuals in a sample with an adverse health condition in period t. It is clear that this group can be partitioned into those whose condition has persisted since period (t-1) and those whose condition is newly-acquired. Now suppose that proportion ρ of the unhealthy group persists each period, and that on average number u acquire the disease. It follows directly that

$$h(t) = \rho h(t-1) + u + \varepsilon(t) \quad , \tag{3}$$

where $\varepsilon(t)$ represents the difference between actual and expected incidence in period t. Embellishments can be added to (3) to make the model more realistic. For example, the number of new contractions u may depend on some set of background variables x(t-1), so that

(3)' $$h(t) = \rho h(t-1) + \beta x(t-1) + \varepsilon(t)$$

under the assumption of linearity.

Thus far, the model has been presented on an aggregate basis. Now define a variable $h_i(t)$ such that $h_i(t) = 1$ if individual i has the adverse health condition in period t, and zero otherwise. It is apparent that $h(t) = \Sigma h_i(t)$. One might also disaggregate the background variables to some level of individual specificity. It should be clear by now that underlying the aggregate dynamic relation (3)' is a family of individual probability equations of the form

(4) $$h_i(t) = \rho h_i(t-1) + \beta x_i(t-1) + \varepsilon_i(t) \quad .$$

Since $h_i(t)$ is defined as a dichotomous variable, and since $\varepsilon(t)$ has zero expectation, it follows that

(4)' $$P[h_i(t) = 1/h_i(t-1), x_i(t-1)] = \rho h_i(t-1) + \beta x_i(t-1) \quad ,$$

which is an elementary linear probability model.

Since the conditioning set for (4)' includes the relevant history of the $h_i(t)$ process, the conditional probability given in (4)' is more accurately described as a transition probability. Moreover, it is clear that for a particular value of x(t), the 2x2 matrix of transition probabilities is easily derived. Denote this matrix by P[x(t)]; then

(5) $$\tilde{p}(t) = \left\{ \prod_{s=1}^{t-1} P[x(t-s)] \right\} \tilde{p}_o[x(0)] \quad ,$$

where $\tilde{p}(t)$ is the vector of probabilities in period t conditional only on the history of background variables [x(t-s), s = 1, 2, ..., t-1], and where $\tilde{p}[x(0)]$ is the vector of initial probabilities. We note that the vector of initial probabilities may also be a function of some set of exogenous variables x(0). By forming the sequence of transition matrices P]x(s)] and then differentiating (5) with respect to x(0), we get the familiar result that the vector of current marginal probabilities $\tilde{p}(t)$ depends on x(0) but to a geometrically decaying degree. In other words, the process is mixing with respect to x(0).

A central aspect of dynamic models is their recognition that the effects of a particular event may vary over time. Typically, an event may have the greatest impact on other events occurring near it in the time dimension and diminished effects on events relatively far in the future. Such is likely to be the case with an individual's genetic endowment, whose impact on health and cognitive development is likely to be strongest relatively close to birth, and then diminish as environmental influences tend to grow. One extreme assumption is to suppose that the genetic endowment affects only the probability vector for initial states, but does not affect the transition probabilities once the initial states are realized. In terms of (5), the genetic endowment would be an element of x(0), but not an element of [x(s), s = 1, 2, ..., t], so that its effects on current marginal probabilities decay geometrically. The corollary, of course, is that the correlation between the individual's genetic endowment and both health and intellectual abilities also decays with time.

One should note that there is a direct correspondence between the dynamic model, given by the transition function (4), and the cross-section model given by the marginal probability function (5), which was generated by (4)'. Alternatively, beginning with a cross-section distribution, we can reconstruct a simple dynamic model. As before, let $h_i(t)$ represent the health status of individual i in period t. For simplicity, let the cross-section distribution be conditional on the (unobserved) genetic endowment (g_i) and a background environmental variable (e_i) for each individual. Restricting our attention to only two periods and neglecting error terms, we have

(6a) $$h_i(1) = a(1)\, g_i + b(1)\, e_i$$

(6b) $$h_i(2) = a(2)\, g_i + b(2)\, e_i \quad .$$

The parameters a(1), b(1), a(2), and b(2) are negative since h(1) and h(2) are negative health indicators, and a more favorable genetic endowment or a more favorable home environment improves health. Define $\lambda_g = a(2)/a(1)$ and $\lambda_e = b(2)/b(1)$. If genetic and environmental effects decay, then $0 < \lambda_g < 1$ and $0 < \lambda_e < 1$. Solve for g_i in (6a) and substitute in (6b) to obtain

(6c) $$h_i(2) = \lambda_g\, h_i(1) + b_2\, (1 - \frac{\lambda_g}{\lambda_e})\, e_i \quad ,$$

which is similar to (4)'. Note that it is plausible to assume that genetic decay exceeds environmental decay ($\lambda_g < \lambda_e$). It follows that the

parameter of e_i in equation (6c)--b(2) $(1 - \frac{\lambda_g}{\lambda_e})$--is negative. Therefore, regardless of the way in which the dynamic model is derived, we expect a favorable environment to improve health in period 2 with health in period 1 held constant.

This simple single-equation model of transition probabilities can be extended without difficulty to vector-valued Markov processes with both discrete and continuous variables. The model in general form can be written

$$(7) \qquad y_i(t) = A\, y_i(t-1) + B\, x_i(t-1) + C\, z_i + \varepsilon_i(t)$$

where $y_i(t)$ now represents a vector of health and cognitive development measures in period t, $x_i(t)$ is a vector of time-varying explanatory variables, z_i is a vector of time-invariant socioeconomic and background variables, and A, B, and C are matrices of coefficients to be estimated. This constitutes the basic linear model which we estimate in Section III.

Two comments regarding (7) should be made. First, non-diagonality of A permits mutual feedback between the processes which are elements of $y_i(t)$. It is, however, impossible to distinguish how this feedback arises, that is, whether it arises because of structural dependencies between variables, behavioral regularities, or a combination of both. We note that testing for the significance of off-diagonal elements of A amounts to a weak test for causality between the elements of $y_i(t)$ in the sense of Granger (1969).

Second, it should be emphasized that the critical assumption underlying the dynamic specification of this model is not that the unobserved

genetic component is unrelated to health status and cognitive development. Rather it is that past observations of these variables already embody genetic factors and that the transition functions depend only on environmental background variables.

II. Empirical Implementation

A. The Data

The model formulated in Section I is estimated using data from Cycles II and III of the U.S. Health Examination Survey (HES) conducted by the National Center for Health Statistics (NCHS). Cycle II is a nationally representative sample of 7,119 noninstitutionalized children aged 6 to 11 years in the 1963-65 period. Cycle III is a nationally representative sample of 6,768 noninstitutionalized youths aged 12 to 17 years in the 1966-70 period. NCHS did not try to reexamine all of the children in Cycle II when the Cycle III sample was selected. Instead, the Cycle III design was such that roughly one-third of the Cycle II sample was reexamined. Specifically, of the total number of children in Cycle II, 2,177 also are in Cycle III. Thus taken together the two cycles represent a longitudinal data set on these 2,177 youths with measures of health and cognitive development of the same youth at two different ages.[9] There are no 17-year-olds in the longitudinal sample and few 16-year-olds; 99 percent of the sample is between the ages of 12 and 15.

The HES data comprise medical histories of each youth provided by the parent, information on family socioeconomic characteristics, birth certificate information, and a school report with data on school performance and classroom behavior provided by teachers or other

school officials. Most important, there are objective measures of health from detailed physical examinations and scores on psychological (including IQ and achievement) tests. The physical examinations were given to the children and youths by pediatricians and dentists employed by the Public Health Service at the time of each cycle of the HES. Similarly, the IQ and achievement tests were given to the children and youths by psychologists employed by the Public Health Service at the time of each cycle.

Our estimates employ panel data for white adolescents who lived with either both of their parents or with their mothers only as of the date of the Cycle II examination. Black adolescents are excluded from the empirical analysis because Edwards and Grossman (1979, and Chapters 2 and 4) have found significant race differences in slope coefficients in cross-sectional research with Cycles II and III. In addition, since the black sample is too small to allow for reliable coefficient estimates, separate estimates for black adolescents are not presented. With two exceptions discussed below, observations are deleted if there are missing data. The final sample size is 1,434. Table 1 contains definitions, means, and standard deviations of each dependent or independent variable in the regressions fitted in Section III. It also contains a notation concerning the source of each variable (parents, birth certificate, physical or dental examination, psychological examination, or school form). The adolescent (current) health and cognitive development measures from Cycle III are denoted by the number 2 at the end of a variable name. The corresponding childhood (lagged) health and cognitive development measures are denoted by the number 1 at the end of a variable name.

TABLE 1
Definition of Variables

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
A. Cognitive Development Measures				
WISC1[c]	103.508	13.924	Youth's IQ as measured by vocabulary and block design subtests of the Wechsler Intelligence Scale for Children, standardized by the mean and standard deviation of four-month age cohorts, in Cycles II and III, respectively	4
WISC2[c]	104.513	13.998		
WRAT1[c]	103.568	12.017	Youth's school achievement as measured by the reading and arithmetic subtests of the Wide Range Achievement Test, standardized by the mean and standard deviation of six-month age cohorts, in Cycles II and III, respectively	4
WRAT2[c]	104.112	13.563		
B. Health Measures				
APERI1[d]	-.055	.792	Periodontal Index, standardized by the mean and standard deviation for one-year age-sex cohorts, in Cycles II and III, respectively	3
APERI2[d]	-.138	.852		
ABN1	.096	.294	Dummy variables that equal one if the physician finds a significant abnormality in examining the youth, in Cycles II and III, respectively	3
ABN2	.188	.391		
HDBP1	.054	.226	Dummy variables that equal one if youth's average diastolic blood pressure is greater than the 95th percentile for the youth's age and sex class, in Cycles II and III, respectively	3
HDBP2	.054	.227		

(continued on next page)

TABLE 1 (continued)

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
OBESE1 OBESE2	.110 .094	.312 .292	Dummy variables that equal one if youth's weight is greater than the 90th percentile for youth's age, sex, and height class, in Cycles II and III, respectively	3
PFGHEALTH1 PFGHEALTH2	.441 .272	.497 .445	Dummy variables that equal one if parental assessment of youth's health is poor, fair or good in Cycles II and III, respectively. Variable equals zero if assessment is very good in Cycle II and very good or excellent in Cycle III; there is no excellent category in Cycle II	1
SCHABS1 SCHABS2[e]	.033 .054	.178 .221	Dummy variables that equal one if youth has been excessively absent from school for health reasons during the past six months, in Cycles II and III, respectively	5
SCHABSUK1	.068	.252	Dummy variable that equals one if information about school absence in Cycle II is not available	5
C. Family and Youth Characteristics[f]				
FEDUCAT[g]	11.310	3.355	Years of formal schooling completed by father	1
MEDUCAT	11.216	2.704	Years of formal schooling completed by mother	1

(continued on next page)

TABLE 1 (continued)

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
FINC	8.060	4.607	Continuous family income (in thousands of dollars) computed by assigning mid-points to the following closed income intervals, $250 to the lowest interval, and $20,000 to the highest interval. The closed income classes are: $500 - $999 $1,000 - $1,999 $2,000 - $2,999 $3,000 - $3,999 $4,000 - $4,999 $5,000 - $6,999 $7,000 - $9,999 $10,000 - $14,999	1
LESS20	3.700	1.813	Number of persons in the household 20 years of age or less	1
MWORKFT MWORKPT	.149 .149	.356 .356	Dummy variables that equal one if the mother works full-time or part-time, respectively; omitted class is mother does not work	1
NEAST MWEST SOUTH	.265 .315 .203	.442 .465 .402	Dummy variables that equal one if youth lives in Northeast, Midwest, or South, respectively; omitted class is residence in West	1

(continued on next page)

TABLE 1 (continued)

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
URB1 URB2 URB3 NURB	.189 .126 .200 .140	.392 .331 .400 .347	Dummy variables that equal one if youth lives in an urban area with a population of 3 million or more (URB1); in an urban area with a population between 1 million and 3 million (URB2); in an urban area with a population less than 1 million (URB3); or in a non-rural and non-urbanized area (NURB); omitted class is residence in a rural area	1
LIGHTA	.008	.091	Dummy variable that equals one if youth's birth weight was under 2,000 grams (under 4.4 pounds)	2
LIGHTB	.054	.227	Dummy variable that equals one if youth's birth weight was equal to or greater than 2,000 grams but under 2,500 grams (under 5.5 pounds)	2
BWUK	.138	.345	Dummy variable that equals one if youth's birth weight is unknown	2
FYPH	.068	.252	Dummy variable that equals one if parental assessment of child's health at one year was poor or fair and zero if it was good	1
BFED	.302	.459	Dummy variable that equals one if the child was breast fed	1
LMAG	.057	.231	Dummy variable that equals one if the mother was less than 20 years old at birth of youth	1

(continued on next page)

TABLE 1 (concluded)

Variable Name	Sample[a] Mean	Sample Standard Deviation	Definition	Source[b]
MAG	.119	.324	Dummy variable that equals one if mother was more than 35 years old at birth of youth	1
NOFATH	.047	.213	Dummy variable that equals one if youth lives with mother only	1
FIRST	.292	.455	Dummy variable that equals one if youth is the first born in the family	1
TWIN	.028	.165	Dummy variable that equals one if youth is a twin	1
FLANG	.110	.312	Dummy variable that equals one if a foreign language is spoken in the home	1
MALE	.522	.500	Dummy variable that equals one if youth is a male	1
AGE	9.712	1.042	Age of youth	1
INTERVAL	42.327	6.404	Number of months between the physical examinations given for the Cycle II survey and the Cycle III survey	3

Footnotes to TABLE 1

[a]The means and standard deviations are for the sample of 1,434 white youths described in the text.

[b]The sources are 1 = parents, 2 = birth certificate, 3 = physical examination, 4 = psychological examination, 5 = school form.

[c]The mean of this variable is not equal to 100 because standardization was done using the entire Cycle II or Cycle III sample rather than the subsample reported here. In particular the mean in excess of 100 reflects the better cognitive development of white youths compared to black youths.

[d]The mean of this variable is not zero because standardization was done using the entire Cycle II or Cycle III sample rather than the subsample reported here. In particular the negative mean reflects the better oral health of white youths compared to black youths.

[e]The mean and standard deviation are based on a subsample of 1,321 youths for whom the school form was available.

[f]All family and youth characteristics are from Cycle II unless otherwise stated.

[g]For youths who were not currently living with their father, father's education was coded at the mean of the sample for which father's education was reported.

assessment of the youth's overall health (PFGHEALTH1, PFGHEALTH2); and excessive school absence for health reasons during the past six months (SCHABS1, SCHABS2). The six health measures are negative correlates of good health, and with the exception of the periodontal index, they are dichotomous variables. In selecting them we are guided by previous research by Edwards and Grossman and by their detailed discussions of the measurement of health in Cycles II and III of the HES (Edwards and Grossman 1979, and Chapters 2 and 4.

Briefly, our choice of health measures is based on several criteria. Clearly, health is a multidimensional concept, and we try to paint as complete a picture as possible of the health of youths in the HES panel. Based on Edwards and Grossman's selection criteria for their cross-sectional study of adolescent health in Cycle III (see Chapter 4), we include at least two indicators of physiological health, obesity and the periodontal index, that (1) reflect deterimental health behaviors or life styles that may persist and create more serious health problems in adulthood and (2) potentially can be modified by endogenous inputs in the health production function such as proper diet, parents' time, and preventive medical care.[11] Appropriate dental care provided by dentists can have a direct impact on periodontal disease. The extent of this disease and obesity reflect basic nutritional factors that can be modified by the appropriate diet. Pediatricians and dentists can make dietary recommendations to parents; thus preventive medical contacts with health personnel and the parents' time are relevant determinants of these outcomes. Moreover, poor oral health and obesity in adolescence are positively correlated with these conditions in adulthood.

B. Measurement of Cognitive Development

Two measures of cognitive development are employed: an IQ measure derived from two subtests of the Wechsler Intelligence Scale for Children(WISC1, WISC2), and a school achievement measure derived from the reading and arithmetic subtests of the Wide Range Achievement Test (WRAT1, WRAT2). Both measures are scaled to have means of 100 and standard deviations of 15 for each age-group (four-month cohorts are used for WISC and six-month cohorts are used for WRAT).[10] The inadequacies of these variables as indexes of overall cognitive development are well known. Nevertheless, they continue to be widely used because they provide a readily obtainable measure that is roughly comparable across a diverse population.

WISC is a common IQ test, similar to (and highly correlated with results from) the Stanford-Binet IQ test (NCHS 1972). The full test consists of twelve subtests, but only two of these were administered in the HES. IQ estimates based on the vocabulary and block design subtests are very highly correlated with those based on all twelve subtests (NCHS 1972). The two WRAT achievement tests used in the HES were found to "... have reasonably good construct validity as judged by their relationship to conventional achievement tests (NCHS 1967b)."

C. Measurement of Health

Six measures of childhood and adolescent health are employed: oral health as reflected by the periodontal index (APERI1, APERI2); obesity (OBESE1, OBESE2); the presence of one or more significant abnormalities as reported by the examining physician (ABN1, ABN2); high diastolic blood pressure (HDBP1, HDBP2); the parent's

Finally, their presence in adolescence previews the propensity of a youth to adopt a whole range of unhealthy practices, such as smoking, excessive alcohol consumption, and the failure to wear seat belts, in adulthood.

Although the health measures other than obesity and the periodontal index are less relevant in terms of the Edwards-Grossman criteria, they convey useful information. There is a lack of consensus among pediatricians concerning the importance of high blood pressure in youths and the appropriate treatment (National Heart, Lung, and Blood Institute's Task Force 1977). Yet it is of interest to study the relationship between this condition, other health conditions, and cognitive development; and to shed light on its potential impacts and on the pure environmental contribution of family background.[11] To a large extent, the health problems identified as significant abnormalities are not subject to medical intervention, but they may impinge on many aspects of current and future well-being. Parental rating of their offspring's health suffers from the defect that it is a subjective indicator: parents with low levels of income and schooling are likely to be dissatisfied with many aspects of their life including the health of their offspring. By holding constant the lagged value of this variable, however, we purge it of some if not all of its subjective element. Excessive school absence due to illness in a six-month period may reflect a natural sequence of adolescent diseases and acute conditions that are self-limiting in nature and do not reflect future health problems. But excessive absence may be detrimental to cognitive development. In addition, youths with serious health problems are more susceptible to acute conditions and are more likely to have them for extended periods of time.[12]

It should be noted that some health indicators are excluded from our empirical estimates because they are very rare or because comparable measures are not available in both cycles of the HES. Abnormal hearing is not studied because its prevalence rate is less than 1 percent in each cycle. Iron-deficiency anemia, as reflected by low hematocrit levels, is excluded because blood tests were administered in Cycle III but not in Cycle II. Abnormal corrected binocular distance vision is available in Cycle III, but it is not available in Cycle II because children who wore eyeglasses were examined without their glasses. Hence, poor vision cannot be employed in the longitudinal analysis.

With regard to the six childhood and adolescent health measures that we do study, the definitions of obesity (OBESE1, OBESE2); high diastolic blood pressure (HDBP1, HDBP2); and parental assessment of health (PFGHEALTH1, PFGHEALTH2) in Table 1 are self-explanatory. The periodontal index (APER1, APERI2) is a good overall indicator of oral health as well as a positive correlate of nutrition (Russell 1956). Kelly and Sanchez (1972, pp. 1-2) describe the periodontal index as follows:

> Every tooth in the mouth ... is scored according to the presence or absence of manifest signs of periodontal disease. When a portion of the free gingiva is inflamed, a score of 1 is recorded. When completely circumscribed by inflammation, teeth are scored 2. Teeth with frank periodontal pockets are scored 6 when their masticatory function is unimpaired and 8 when it is impaired. The arithmetic average of all scores is the individual's [periodontal index], which ranges from a low of 0.0 (no inflammation or periodontal pockets) to a high of 8.0 (all teeth with pockets and impaired function).

Due to the significant age and sex trends in the periodontal index, our measure is computed as the difference between the adolescent's actual index and the mean index for his or her age-sex group divided by the standard deviation for that age-sex group. Higher values of APERI denote poorer values of oral health. Note that poor oral health is one of the few health problems for which well-defined continuous measures of poor health have been constructed.

Significant abnormalities (ABN1, ABN2) include heart disease; neurological, muscular, or joint conditions; other major diseases; and in Cycle III only otitis media. This last condition constitutes a relatively small percentage (approximately 1 percent) of all reported abnormalities in Cycle III. Excessive school absence due to illness in the past six months (SCHABS1, SCHABS2) is taken from information provided by the child's or youth's school. There is no school form for roughly 7 percent of the sample. Since excessive absence due to illness is the only variable taken from the school form, a dummy variable that identifies youths with missing Cycle II school forms (SCHABSUK1) is included in all regression equations as an independent variable. Youths without a Cycle III school form are eliminated from the empirical analysis only when SCHABS2 is the dependent variable.

D. Measurement of Other Variables

In addition to lagged (childhood) health and cognitive development, each regression contains the set of family and youth characteristics defined in part C of Table 1. We will not discuss the role of each member of this set because detailed treatments can be found in previous research by Edwards and Grossman (1979, and Chapters 2 and 4).

Instead, we conclude this section with a few general comments on several family and youth characteristics.

We are particularly concerned with the effects of three family background or home environmental variables: mother's schooling (MEDUCAT), father's schooling (FEDUCAT), and family income (FINC). Parents' schooling reflects their efficiency in the production of their offspring's health and cognitive development, while family income reflects command over resources. In general we do not discuss the effects of other background variables in this paper, but it should be realized that all effects that we do discuss control for (hold constant) the impacts of these variables.[13] With the exception of the number of months between the date on which a youth was examined in Cycle II and the date on which he was examined in Cycle III (INTERVAL), all family and youth characteristics are taken from Cycle II. Thus, we relegate to future research a study of the effects of changes over time in background variables such as family income and mother's labor force status.

The youth's age as of the Cycle II exam (AGE) and sex (MALE) are included in regressions in which the dependent variable is not adjusted for age and sex. Hence, they are employed in regressions that explain the presence of significant abnormalities, parental assessment of health, and excessive school absence due to illness. Sex but not age is included in regressions that explain the two cognitive development measures.[14]

III. Empirical Results

Ordinary least squares multiple regression equations for the dependent variables WISC2, WRAT2, APERI2, ABN2, HDBP2, PFGHEALTH2, OBESE2, and SCHABS2 are given in Tables A-1 through A-8 in the appendix. Since the six adolescent health measures are negative correlates of good health, negative effects of family background and lagged cognitive development in the health equations reflect factors associated with better health outcomes. Similarly, positive coefficients of lagged health in the current health equations signify that poor health in childhood is associated with poor health in adolescence. Finally, negative coefficients of lagged health in the current cognitive development equations mean that poor health in childhood reduces cognitive development in adolescence. Although five of the eight dependent variables are dichotomous, the method of estimation is ordinary least squares. Preliminary investigation revealed almost no differences between ordinary least squares estimates and dichotomous logit estimates obtained by the method of maximum likelihood. Except when the periodontal index is the dependent variable, each fitted health equation can be interpreted as a linear probability function in which the regression coefficient of a given continuous variable shows the change in the conditional probability of poor adolescent health for a one-unit change in the independent variable. The regression coefficient of a dichotomous variable shows the difference in conditional probabilities between the two groups in question. The evidence with regard to causal priorness is discussed in the first part of this section. The estimated effects of family background are discussed in the second part.

A. Causal Priorness

Table 2 contains the 8 by 8 matrix of lagged coefficients from the regression equations. The main diagonal elements of the matrix are the own-lagged effects or the regression coefficients of the lagged dependent variable. For example, the regression coefficient of WISC1 in the WISC2 equation is .60, and the coefficient of HDBP1 in the HDBP2 equation is .17. The off-diagonal elements of the matrix provide information with regard to mutual feedback between health and cognitive development, mutual feedback between various health conditions, and mutual feedback between IQ (WISC) and school achievement (WRAT).

Each of the eight own-lagged effects is positive and statistically significant at all conventional levels of confidence.[15] The coefficients range from a high of .73 in the case of WRAT to a low of .15 in the case of ABN.[16] These coefficients are not without interpretation. Coefficients close to one serve as indicators that a particular process is relatively slow frequency or slowly changing. Coefficients close to zero indicate a higher frequency process. For the five dichotomous health measures, the own-lagged coefficients can be interpreted as the degree of persistence in the particular aspect of health in question. This can be seen immediately since the lagged coefficient is the difference between the expected conditional probability of an adolescent health condition given that the same condition was present in childhood and the conditional probability given that the condition was absent in childhood. Obesity is the most persistent of the dichotomous measures: obese children have approximately 50 percentage point higher probabilities of being obese adolescents than non-obese children.

TABLE 2

Regression Coefficients of Lagged Health and Lagged Cognitive Development[a]

Current \ Lagged	WISC1	WRAT1	APERI1	ABN1	HDBP1	PFGHEALTH1	OBESE1	SCHABS1
WISC2	.603 (27.35)	.231 (9.47)	-.164 (-0.54)	-1.619 (-2.15)	-1.791 (-1.82)	.388 (0.84)	.946 (1.33)	-.650 (-0.53)
WRAT2	.192 (9.79)	.728 (33.52)	-.073 (-0.27)	-.204 (-0.30)	-.740 (-0.85)	-.341 (-0.82)	.421 (0.66)	-.699 (-0.64)
APERI2	-.004 (-2.25)	-.005 (-2.35)	.340 (12.16)	-.005 (-0.07)	-.195 (-2.15)	.039 (0.90)	.114 (1.73)	.146 (1.28)
ABN2	-.001 (-0.98)	-.002 (-2.00)	.020 (1.41)	.146 (4.16)	.042 (0.91)	.001 (0.03)	.049 (1.47)	.064 (1.11)
HDBP2	-.001 (-1.11)	-.0003 (-0.51)	-.003 (-0.41)	.030 (1.47)	.169 (6.38)	-.010 (-0.78)	.096 (4.97)	.033 (1.00)
PFGHEALTH2	-.001 (-0.92)	-.003 (-2.76)	.019 (1.22)	.139 (0.37)	.043 (0.86)	.243 (10.43)	.019 (0.52)	.096 (1.54)
OBESE2	.0001 (0.08)	-.001 (-1.45)	-.014 (-1.60)	-.020 (-0.91)	.013 (0.43)	-.016 (-1.15)	.512 (24.28)	.007 (0.18)
SCHABS2	.0002 (0.34)	-.001 (-1.78)	.004 (0.49)	.011 (0.49)	.009 (0.34)	.045 (3.45)	.020 (1.03)	.159 (4.60)

[a]t-ratios are in parentheses. The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test. Source: Appendix Tables A-1 through A-8.

The most striking message in Table 2 is that causality from cognitive development to health is much more important than causality from health to cognitive development. None of the six health measures is statistically significant in the WRAT equation. Moreover, the null hypothesis that no member of the set of six health variables has a non-zero effect on WRAT2 is accepted at the 5 percent level.[17] In the WISC2 equation, only two of the six health coefficients are significant at the 5 percent level. Specifically, youths who had one or more significant abnormalities in childhood have WISC scores that are approximately 2 points lower than other youths. A similar differential emerges when the WISC scores of youths with high diastolic blood pressure in childhood are compared to those of other youths.

Although only two of the twelve health coefficients are significant in the cognitive development equations, five of the twelve cognitive development coefficients are significant at the 5 percent level in the health equations. Put differently, at least one of the two cognitive development variables is significant in four of the six health equations, and both are significant in the periodontal index equation. Given the high correlation between WISC1 and WRAT1 ($r = .63$), this is an impressive finding. All of the significant cognitive development coefficients have the predicted negative sign. In particular, an increase in cognitive development in childhood reduces the periodontal index, the probability of at least one significant abnormality, the probability of a parental rating of health that is worse than very good, and the probability of excessive school absence due to illness in adolescence. Moreover, the negative effect of school achievement on obesity is

statistically significant at the 7.5 percent level although not at the 5 percent level. Finally, both WISC and WRAT lower the probability of high blood pressure, although neither regression coefficient is significant even at the 10 percent level.[18]

With regard to other causal relationships in Table 2, there is evidence of mutual feedback between IQ and school achievement. There is also evidence that obesity in childhood causes poorer oral health and high blood pressure in adolescence. Moreover, a parental rating of health in childhood as poor, fair, or good (as opposed to very good) is associated with excessive school absence due to illness in adolescence. Finally, there is one seemingly "perverse" and statistically significant relationship in the table: high blood pressure in childhood is associated with better oral health in adolescence.

A full explanation of the more interesting causal relationships in Table 2 is both difficult and challenging. As we pointed out in Section I, strictly speaking it is impossible to distinguish how feedbacks arise in our simple dynamic model. Nevertheless, a number of relevant factors can be mentioned, partly as hypotheses for future research. The causal effects of childhood health on adolescent health probably reflect structural dependencies rather than behavioral regularities. To be specific, the causal effect of obesity on high blood pressure can be traced to a physiological relationship. Physicians indicate that obesity is a risk factor in the incidence of high blood pressure in adolescents, although their supporting evidence relies on cross-sectional data (see National Heart, Lung, and Blood Institute's Task Force 1977). The causal priorness of obesity in poor oral health outcomes can be traced to basic nutritional factors that are known to

be associated with periodontal disease (Russell 1956). That is, obese children are likely to consume foods that are detrimental to oral health.

Causal effects of childhood cognitive development on adolescent health can be plausibly attributed to behavioral regularities as opposed to structural dependencies. Adolescents are more active producers of their own health than children. Thus the adolescent's time and efficiency are important variables in the health production function. For example, more intelligent youths are more likely to be able to follow doctors' and parents' instructions, to have general information about nutrition, and to be willing and able to acquire medical information from published materials. Consequently, one would expect such youths to have higher health levels than their less intelligent peers. It should be noted that an individual's cognitive development, measured by IQ or by achievement tests, is perhaps the most important determinant of the number of years of formal schooling that he ultimately completes.[19] Therefore, the causal effect of cognitive development on adolescent health can be viewed as the early forerunner of the positive impact of schooling on good health for adults in the United States reported by Grossman (1975), Shakotko (1977), and others.[20]

Although feedback from childhood health to adolescent cognitive development is not as strong as feedback from cognitive development to health, we do report important effects for high blood pressure and significant abnormalities. Moreover, Grossman (1975) finds that years of college completed is negatively related to poor health status in high school. In addition, Leveson, Ullman, and Wassall (1969) cite a

number of studies indicating that about 5 to 7 percent of persons dropping out of high school in the United States have done so primarily because of illness. Our findings and these call attention to the existence of a continuing interaction between health and intellectual development over the life cycle.

B. Family Background Effects

Table 3 contains regression coefficients of mother's schooling (MEDUCAT), father's schooling (FEDUCAT), and family income (FINC), in the eight cognitive development and health functions. The estimates in the first three columns, labeled cross-sectional coefficients, are taken from multiple regressions that control for all family and youth characteristics in Table 1 but exclude all lagged (childhood) cognitive development and health measures. The estimates in the last three columns, labeled dynamic coefficients, are taken from multiple regressions that include all lagged cognitive development and health measures as well as family and youth characteristics. The first set of estimates shows background effects computed in a cross-section. The second set shows background effects estimated in a dynamic context which controls for initial levels of cognitive development and health. As we argued in Section I, the dynamic estimates are free of genetic-bias; they indicate the pure contribution of the home environment to cognitive development and health outcomes in adolescence.

When the lagged variables are omitted from the cognitive development functions, all six family background coefficients are positive and statistically significant. When the lagged variables are included,

TABLE 3

Regression Coefficients of Parent's Schooling and Family Income[a,b]

Dependent Variable \ Independent Variable	Cross-Section Coefficients			Dynamic Coefficients		
	MEDUCAT	FEDUCAT	FINC	MEDUCAT	FEDUCAT	FINC
WISC2	.986	.904	.288	.146	.207	.135
	(6.19)	(6.80)	(3.33)	(1.32)	(2.24)	(2.27)
WRAT2	.942	.805	.271	.177	.136	.103
	(6.03)	(6.18)	(3.20)	(1.79)	(1.65)	(1.94)
APERI2	-.039	-.019	0.005	-.023	-.006	.0001
	(-3.67)	(-2.17)	(-0.91)	(-2.25)	(-0.75)	(0.00)
ABN2	-.002	-.005	.004	.003	-.003	.005
	(-0.36)	(-1.26)	(1.60)	(0.51)	(-0.65)	(1.83)
HDBP2	-.005	.002	-.001	-.003	.003	-.001
	(-1.84)	(0.66)	(-0.33)	(-0.89)	(1.07)	(-0.53)
PFGHEALTH2	-.015	-.012	-.007	-.009	-.006	-.001
	(-2.71)	(-2.47)	(-2.21)	(-1.69)	(-1.23)	(-0.47)
OBESE2	-.012	.00002	.001	-.005	.001	.0004
	(-3.19)	(0.00)	(0.55)	(-1.42)	(0.53)	(0.21)
SCHABS2	-.010	.003	-.002	-.008	.004	-.001
	(-3.11)	(1.09)	(-1.32)	(-2.46)	(1.35)	(-0.66)

[a] t-ratios are in parentheses. The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test. The cross-sectional coefficients are taken from multiple regressions that contain all family and youth characteristics. The dynamic coefficients are taken from multiple regressions that contain all variables.

[b] Source: Appendix Tables A-1 through A-8 (dynamic estimates only).

only the coefficient of mother's schooling in the WISC2 regression becomes insignificant. The magnitudes of the dynamic family background effects are, however, much smaller than the magnitudes of the cross-sectional effects. To be precise, the ratios of dynamic coefficients to the corresponding cross-sectional coefficients range from .15 in the case of mother's schooling in the WISC2 equation to .47 in the case of family income in the same equation.

In the cross-sectional health regressions, all six regression coefficients of mother's schooling are negative, and only the coefficient in the abnormality equation is not significant. Father's schooling has negative and significant coefficients in two equations alone: those for the periodontal index and parental assessment of health. In the latter equation but nowhere else the family income effect is negative and significant. The eight statistically significant background effects are reduced in absolute value when adolescent health and cognitive development are included in the regressions. The ratios of the dynamic coefficients to the corresponding cross-sectional coefficients range from .14 in the case of family income in the PFGHEALTH2 equation to .80 in the case of mother's schooling in the SCHABS2 equation. Moreover, there are only three statistically significant dynamic coefficients at the 5 percent level: those belonging to mother's schooling in the APERI2, PFGHEALTH2, and SCHABS2 equations. Note that the negative effect of mother's schooling on obesity is significant at the 8 percent level but not at the 5 percent level.

A clear message in Table 3 is that the dynamic effects of family background on cognitive development and health are much smaller than

the cross-sectional effects. It is important to realize that this finding does not mean that family background is an unimportant determinant of adolescent health and cognitive development. The dynamic effects are short-run effects in the sense that they hold constant the lagged values of health and cognitive development. Since these lagged values depend on family background, the cumulative or long-run impacts of family background are likely to exceed the dynamic or short-run impacts. To be precise, if cross-lagged effects are ignored, a full representation of the dynamic health process that we study is

$$h(1) = \alpha g + b(1)\, e \tag{8}$$

$$h(t) = a(t)\, h(t-1) + b(t)\, e,\ t = 2, \ldots, n \tag{9}$$

In these equations h(t) is health at age t, g is genetic endowment, and e is the home environment. Solving recursively, one obtains

$$h(t) = \left[\alpha \prod_{i=1}^{t} a(t)\right] g + \left[b(t) + \sum_{i=1}^{t-1} b(i) \prod_{j=t-1}^{i} a(j)\right] e \tag{10}$$

The parameter of e in equation (10) is the cumulative environmental effect. If the b(i) all have the same sign, the long-run parameter unambiguously exceeds b(t) in absolute value. The long-run effect may be larger or smaller than the cross-sectional effect obtained by regressing h(t) on e with g omitted. Since it is plausible that environmental effects decay with age and since we have only two observations on a given individual, we do not compute cumulative effects.[21]

Another message in Table 3 is that mother's schooling dominates both family income and father's schooling as a statistically significant determinant of favorable adolescent health outcomes. The unimportance of family income complements previous findings concerning the health of children and adolescents by Edwards and Grossman (1980, forthcoming). A similar comment applies to the magnitudes of the schooling coefficients. Given that most child and adolescent care is provided by the mother, the finding that the effect of mother's schooling exceeds that of father's schooling is evidence in favor of the importance of nurture as opposed to nature in health outcomes. To be sure, the schooling differentials must be interpreted with caution because the difference between schooling coefficients in a given health equation never is statistically significant. In part this is due to the high correlation between mother's schooling and father's schooling ($r = .62$). Despite this high correlation, 83 percent of the cross-sectional mother's schooling coefficients are statistically significant, and 50 percent of the dynamic coefficients are significant. The corresponding figures for father's schooling are 29 percent and 0 percent. This is suggestive of a larger impact of mother's schooling on health outcomes vis-a-vis father's schooling.

In the cognitive development equations, family income and father's schooling are as important as, if not more important than, mother's schooling. We speculate that this difference between the role of family background in cognitive development and its role in health may arise because adolescent cognitive development is produced primarily in the market (in school), while adolescent health is produced primarily in

the home. Thus in the cognitive development equations, the variables that reflect command over goods and services--family income and father's schooling, a positive correlate of permanent income--are significant predictors.[22] On the other hand, in the health equations, nonmarket productivity, measured by mother's schooling, dominates command over goods and services as a significant predictor.

In part A of this section, we reported that feedbacks from cognitive development to health is much more important than feedback from health to cognitive development. Therefore, cognitive development is one mechanism via which family background can influence adolescent health. To gauge the importance of this mechanism, we have estimated the effects of family background on the six adolescent health measures from multiple regressions that include all lagged health variables but exclude WISC1 and WRAT1. These estimates are presented in Table 4. Clearly, the coefficients in Table 4 are larger in absolute value than the corresponding dynamic coefficients in Table 3. Moreover, the coefficient of mother's schooling in the obesity equation, although numerically the same as in Table 3, becomes significant at the 5 percent level; so that four of the six mother's schooling effects are significant. The identification of a plausible mechanism via which family background in general and mother's schooling in particular influence adolescent health has increased our confidence that the impact of schooling represents a behavioral effect as opposed to a genetic effect or a statistical artifact.

To summarize, the results in Tables 3 and 4 support the contention that "nurture" matters. The short-run effects of mother's schooling on

TABLE 4

Coefficients of Parents' Schooling and Family Income in Health Regressions[a]

Dependent Variable \ Independent Variable	MEDUCAT	FEDUCAT	FINC
APERI2	-.032 (-3.15)	-.014 (-1.64)	-.002 (-0.30)
ABN2	-.0001 (-0.00)	-.005 (-1.23)	.005 (1.62)
HDBP2	-.004 (-1.24)	.002 (0.76)	-.001 (-0.64)
PFGHEALTH2	-.013 (-2.36)	-.009 (-1.94)	-.002 (-0.73)
OBESE2	-.005 (-1.69)	.001 (0.28)	.0002 (0.10)
SCHABS2	-.008 (-2.72)	.003 (1.14)	-.001 (-0.75)

[a]t-ratios are in parentheses. The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test. The equation contains all family and youth characteristic variables and all lagged health variables but excludes WISC1 and WRAT1.

obesity, oral health, school absence due to illness, and parental evaluation of health are significant; and the long-run effects of mother's schooling on these health indicators and others are probably more dramatic.

One other finding from the empirical analysis is worth mentioning. The dynamic regression coefficients of birth weight, mother's age at the birth of the youth, and parental assessment of the youth's health in the first year of his life are almost never statistically significant (see Tables A-1 through A-8). These variables are proxy measures of genetic endowment. Therefore, these results support the constraint in our model that adolescent health and cognitive development should not depend on genetic endowment with childhood health and cognitive development held constant.

IV. Summary and Implications

The purposes of this study have been to determine the directions of causality associated with the relationship between the health and cognitive development of adolescents and to examine the extent to which the effects of family background on health and cognitive development are due to nurture as opposed to nature. The study contains two basic findings. First, there is feedback from health to cognitive development and from cognitive development to health, but the latter of these relationships is stronger. Second, dynamic family background effects are smaller than their cross-sectional counterparts, but a number of the estimated effects are statistically significant.

The first finding calls attention to the existence of a continuing interaction between health and cognitive development over the life cycle.

In addition, it can be viewed as the early forerunner of the positive impact of schooling on the health of adults. The second finding suggests that nurture "matters" in cognitive development and health outcomes. In particular, mother's schooling is singled out as the crucial component of the home environment in adolescent health outcomes. This is an especially strong result because in the words of Keniston and the Carnegie Council on Children: "Doctors do not provide the bulk of health care for children; families do (1977, p. 179)." Since the mother spends more time in household production than the father, her characteristics should be the dominant factor in outcomes that are determined to a large extent in the home. The importance of mother's schooling in obesity and oral health is notable because these are outcomes that are neither irreversible or self-limiting. Instead, they can be modified by inputs of dental care, medical care, proper diet, and parents' time.

The two findings interact with each other. Cognitive development in childhood has a positive effect on health in adolescence, and cognitive development in childhood is positively related to mother's schooling. Both findings imply that the health of adults is heavily dependent upon their home environment as youths. They also imply that public policies aimed at children's and adolescent's health must try to offset the problems encountered by offspring of mothers with low levels of schooling. In particular, they should try to improve the skills of uneducated mothers in their capacity as the main provider of health care for their offspring.

TABLE A-1

Ordinary Least Squares Regression of WISC2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	.207	2.24	FIRST	.960	1.77
MEDUCAT	.146	1.32	TWIN	-2.177	-1.58
FINC	.135	2.27	FLANG	.643	0.85
LESS20	.138	0.97	MALE	2.674	5.95
MWORKPT	.965	1.53	AGE	-	-
MWORKFT	-.390	-0.61	INTERVAL	-.004	-0.09
NEAST	4.503	6.44	WISC1	.603	27.35
MWEST	2.297	3.64	WRAT1	.231	9.47
SOUTH	1.189	1.60	APERI1	-.164	-0.54
URB1	-1.428	-2.09	HDBP1	-1.791	-1.82
URB2	-.488	-0.65	PFGHEALTH1	.388	0.84
URB3	-.729	-1.15	OBESE1	.946	1.33
NURB	-.182	-0.25	SCHABS1	-.650	-0.53
LIGHTA	4.636	1.78	SCHABSUK1	.959	1.10
LIGHTB	.291	0.27	ABN1	-1.619	-2.15
BWUK	.235	0.34			
FYPH	-1.597	-1.81	CONSTANT	8.810	
BFED	1.174	2.36			
LMAG	.830	0.83	Adj. R^2	.658	
HMAG	.055	0.08			
NOFATH	1.908	1.81	F[b]	79.74	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

TABLE A-2

Ordinary Least Squares Regression of WRAT2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	.136	1.65	FIRST	.125	0.26
MEDUCAT	.177	1.79	TWIN	-.950	-0.77
FINC	.103	1.94	FLANG	.285	0.42
LESS20	.067	0.53	MALE	-.739	-1.85
MWORKPT	.794	1.42	AGE	-	-
MWORKFT	.142	0.25	INTERVAL	-.058	-1.66
NEAST	4.089	6.57	WISC1	.192	9.79
MWEST	2.404	4.29	WRAT1	.728	33.52
SOUTH	1.526	2.30	APERI1	-.073	-0.27
URB1	-.874	-1.44	HDBP1	-.740	-0.85
URB2	.295	0.44	PFGHEALTH1	-.341	-0.82
URB3	1.799	3.19	OBESE1	.421	0.66
NURB	-.028	-0.04	SCHABS1	-.699	-0.64
LIGHTA	1.560	0.67	SCHABSUK1	.265	0.34
LIGHTB	.408	0.43	ABN1	-.204	-0.30
BWUK	-.829	-1.37			
FYPH	-.684	-0.87	CONSTANT	4.336	
BFED	1.081	2.45			
LMAG	.032	0.03	Adj. R^2	.712	
HMAG	1.334	2.16			
NOFATH	-.355	-0.38	F[b]	101.983	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

TABLE A-3

Ordinary Least Squares Regression of APERI2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	-.006	-0.75	FIRST	.042	0.83
MEDUCAT	-.023	-2.25	TWIN	.176	1.39
FINC	.0001	0.00	FLANG	-.047	-0.67
LESS20	.023	1.77	MALE	-	-
MWORKPT	.001	0.00	AGE	-	-
MWORKFT	-.004	-0.07	INTERVAL	-.026	-7.03
NEAST	-.194	-3.02	WISC1	-.004	-2.25
MWEST	-.162	-2.79	WRAT1	-.005	-2.35
SOUTH	.012	0.17	APERI1	.340	12.16
URB1	.117	1.86	HDBP1	-.195	-2.15
URB2	.001	0.00	PFGHEALTH1	.039	0.90
URB3	-.023	-0.40	OBESE1	.114	1.73
NURB	.055	0.84	SCHABS1	.146	1.28
LIGHTA	-.371	-1.55	SCHABSUK1	.016	0.19
LIGHTB	-.078	-0.80	ABN1	-.005	-0.07
BWUK	.035	0.56			
FYPH	.067	0.82	CONSTANT	2.268	
BFED	-.072	-1.57			
LMAG	-.008	-0.09	Adj. R^2	.165	
HMAG	-.017	-0.26			
NOFATH	.106	1.39	F[b]	9.34	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

TABLE A-4

Ordinary Least Squares Regression of ABN2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	-.003	-0.65	FIRST	-.013	-0.53
MEDUCAT	.003	0.51	TWIN	-.069	-1.08
FINC	.005	1.83	FLANG	-.029	-0.82
LESS20	-.010	-1.48	MALE	.006	0.27
MWORKPT	-.003	-0.12	AGE	.015	1.44
MWORKFT	.012	0.40	INTERVAL	-.003	-1.40
NEAST	-.057	-1.75	WISC1	-.001	-0.98
MWEST	.014	0.47	WRAT1	-.002	-2.00
SOUTH	.096	2.77	APERI1	.020	1.41
URB1	.077	2.42	HDBP1	.042	0.91
URB2	-.058	-1.65	PFGHEALTH1	.001	0.03
URB3	.052	1.77	OBESE1	.049	1.47
NURB	.006	0.17	SCHABS1	.064	1.11
LIGHTA	-.013	-0.10	SCHABSUK1	.006	0.15
LIGHTB	-.027	-0.54	ABN1	.146	4.16
BWUK	-.012	-0.38			
FYPH	.143	3.45	CONSTANT	.430	
BFED	.040	1.74			
LMAG	-.036	-0.76	Adj. R^2	.043	
HMAG	.030	0.92			
NOFATH	.007	0.14	F[b]	2.78	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

TABLE A-5

Ordinary Least Squares Regression of HDBP2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	.003	1.07	FIRST	.013	0.87
MEDUCAT	-.003	-0.89	TWIN	.034	0.92
FINC	-.001	-0.53	FLANG	.035	1.73
LESS20	-.001	-0.33	MALE	-	-
MWORKPT	-.009	-0.50	AGE	-	-
MWORKFT	-.007	-0.41	INTERVAL	-.002	-1.62
NEAST	.014	0.77	WISC1	-.001	-1.11
MWEST	.021	1.22	WRAT1	-.0003	-0.51
SOUTH	.049	2.43	APERI1	-.003	-0.41
URB1	.041	2.22	HDBP1	.169	6.38
URB2	.030	1.49	PFGHEALTH1	-.010	-0.78
URB3	.032	1.84	OBESE1	.096	4.97
NURB	.011	0.56	SCHABS1	.033	1.00
LIGHTA	.012	0.17	SCHABSUK1	.018	0.75
LIGHTB	-.005	-0.17	ABN1	.030	1.47
BWUK	.026	1.39			
FYPH	.0004	0.00	CONSTANT	.168	
BFED	.003	0.24			
LMAG	-.019	-0.71	Adj. R^2	.051	
HMAG	.015	0.82			
NOFATH	.027	0.96	F[b]	3.28	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

TABLE A-6

Ordinary Least Squares Regression of PFGHEALTH2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	-.006	-1.23	FIRST	.008	0.29
MEDUCAT	-.009	-1.69	TWIN	-.140	-2.02
FINC	-.001	-0.47	FLANG	-.029	-0.77
LESS20	.009	1.20	MALE	-.002	-0.10
MWORKPT	.031	0.97	AGE	.010	0.92
MWORKFT	-.015	-0.48	INTERVAL	.001	0.31
NEAST	-.024	-0.69	WISC1	-.001	-0.92
MWEST	-.046	-1.44	WRAT1	-.003	-2.76
SOUTH	-.025	-0.66	APERI1	.019	1.22
URB1	.022	0.65	HDBP1	.043	0.86
URB2	.007	0.19	PFGHEALTH1	.243	10.43
URB3	.039	1.23	OBESE1	.019	0.52
NURB	.048	1.34	SCHABS1	.096	1.54
LIGHTA	-.239	-1.82	SCHABSUK1	-.052	-1.20
LIGHTB	.103	1.91	ABN1	.139	0.37
BWUK	-.015	-0.45			
FYPH	.153	3.45	CONSTANT	.639	
BFED	-.020	-0.81			
LMAG	.047	0.94	Adj. R^2	.147	
HMAG	-.002	-0.04			
NOFATH	.062	1.16	F[b]	7.86	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

TABLE A-7

Ordinary Least Squares Regression of OBESE2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	.001	0.53	FIRST	-.002	-0.15
MEDUCAT	-.005	-1.42	TWIN	-.013	-0.32
FINC	.0004	0.21	FLANG	-.001	-0.06
LESS20	-.007	-1.73	MALE	-	-
MWORKPT	.008	0.45	AGE	-	-
MWORKFT	.027	1.41	INTERVAL	-.001	-1.07
NEAST	-.009	-0.45	WISC1	.0001	0.08
MWEST	-.011	-0.59	WRAT1	-.001	-1.45
SOUTH	-.012	-0.56	APERI1	-.014	-1.60
URB1	-.021	-1.05	HDBP1	.013	0.43
URB2	-.029	-1.28	PFGHEALTH1	-.016	-1.15
URB3	-.011	-0.61	OBESE1	.512	24.28
NURB	-.007	-0.31	SCHABS1	.007	0.18
LIGHTA	-.035	-0.46	SCHABSUK1	-.034	-1.34
LIGHTB	.020	0.64	ABN1	-.020	-0.91
BWUK	-.005	-0.26			
FYPH	.024	0.91	CONSTANT	.284	
BFED	-.014	-0.99			
LMAG	-.016	-0.53	Adj. R^2	.314	
HMAG	.058	2.81			
NOFATH	-.022	-0.70	F[b]	20.30	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

TABLE A-8

Ordinary Least Squares Regressions of SCHABS2[a]

Independent Variable	Regression Coefficient	t-ratio	Independent Variable	Regression Coefficient	t-ratio
FEDUCAT	.004	1.35	FIRST	-.005	-0.32
MEDUCAT	-.008	-2.46	TWIN	-.017	-0.44
FINC	-.001	-0.66	FLANG	-.004	-0.20
LESS20	-.005	-1.14	MALE	-.019	-1.52
MWCRKPT	-.014	-0.79	AGE	.017	2.69
MWORKFT	.008	0.43	INTERVAL	.001	0.76
NEAST	-.005	-0.26	WISC1	.0002	0.34
MWEST	-.010	-0.56	WRAT1	-.001	-1.78
SOUTH	.004	0.20	APERI1	.004	0.49
URB1	-.009	-0.47	HDBP1	.009	0.34
URB2	-.020	-0.96	PFGHEALTH1	.045	3.45
URB3	-.001	-0.06	OBESE1	.020	1.03
NURB	-.057	-2.87	SCHABS1	.159	4.60
LIGHTA	-.078	-1.09	SCHABSUK1	-.015	-0.62
LIGHTB	-.005	-0.17	ABN1	.011	0.49
BWUK	-.015	-0.77			
FYPH	.018	0.73	CONSTANT	.045	
BFED	-.013	-0.91			
LMAG	-.038	-1.34	Adj. R^2	.043	
HMAG	-.026	-1.31			
NOFATH	.024	0.81	F[b]	2.63	

[a]The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

[b]Statistically significant at the 1 percent level of significance.

FOOTNOTES

[1]For a partial survey of this literature, see Grossman (1975) and Edwards and Grossman (1979).

[2]In Chapter 4, Edwards and Grossman provide evidence that points to the importance of nurture, but they do not employ the longitudinal framework and panel data that are used in this study.

[3]For recent studies of cognitive development in a longitudinal framework, see for example, Hanushek (1971), Maynard (1977), Ritzen and Winkler (1977), Sumers and Wolfe (1977), and Wolfe (in progress). None of these studies deals with the issue of two-way causality between cognitive development and health.

[4]See Griliches (1974).

[5]See, for example, the study of brothers by Chamberlain and Griliches (1974).

[6]See, for example, the study of earning mobility by Lillard and Willis (1978) and the study of employment patterns by Heckman (1978).

[7]The consistency of both the random and fixed effects models has been shown by Amemiya (1969) and Maddala (1971). Amemiya goes on to prove consistency of the maximum likelihood estimator. In most cases, consistency required $T \to \infty$, where T is the number of observations per individual.

[8]Amemiya (1969) shows that consistency of this GLS/random effects procedure is maintained when σ_λ^2 is chosen arbitrarily for use in $\hat{\Sigma}$, as long as it is chosen to be positive. Again, we require $T \to \infty$. The difficulty is that for finite T, the biases in the random effects estimates may still be large.

[9]Cycles II and III are described in detail in NCHS (1967a) and (1969), respectively. For the information of the reader, Cycle I of the HES is a sample of adults ages 18 to 79 in the period 1960-62.

[10]WISC and WRAT scores sometimes are adjusted for sex as well as for age, but the variables that we use are not sex-adjusted.

[11]The measures of high blood pressure in Cycle III have been criticized because their average values are higher than in comparable samples of adolescents (NCHS 1977). There is no reason to believe, however, that the errors of measurement are correlated with the independent variables in our regression equations. Moreover, as we report in Section III there is a strong relationship between high diastolic blood pressure in

adolescence and childhood. This strengthens our confidence in the reliability of the Cycle III measure at least for the members of the longitudinal panel.

[12]In Chapter 4, Edwards and Grossman exclude some measures in their study of adolescent health that we include here. This is because they examine adolescent health in a cross-sectional context, focus on the effects of preventive medical care, and do not consider the relationship between health and cognitive development.

[13]Since birth certificates are missing for 14 percent of the sample and since birth weight (LIGHTA, LIGHTB) is the only variable taken from the birth certificate, we do not delete these observations. Instead, we include a dummy variable that identifies youths with missing birth certificates (BWUK) in the regression.

[14]The periodontal index and the two cognitive development measures are continuous variables. In these cases we have experimented with the raw score as the dependent variable in a multiple regression that includes age in Cycle II, the square of age, the time interval between the Cycle II and III examinations, the square of the interval, the product of age and the interval, and a dummy variable for male adolescents in the set of independent variables. The results obtained (not shown) with respect to family background, lagged health, and lagged cognitive development effects are similar to those reported in Section III.

[15]Statements concerning statistical significance in the text are based on one-tailed tests except when the direction of the effect is

unclear on a priori grounds or when the estimated effect has the "wrong sign." In the latter cases two-tailed tests are used.

[16]If the dynamic processes that we study have the same structures over time and if cross-lagged effects are ignored, they all have stable long-run solutions. To be specific, let

$$h(t) = ah(t-1) + be \quad ,$$

where h(t) is health at age t, h(t-1) is health at age t-1, and e is the home environment. The long-run solution, obtained by setting h(t) equal to h(t-1), is

$$h(t) = \left(\frac{b}{1-a}\right) e \quad .$$

This is a stable solution provided a is positive and smaller than one.

[17]The F statistic is .36.

[18]The null hypothesis that no member of the set of cognitive development variables has a nonzero effect on HDBP2 is accepted at the 5 percent level (F = 1.54).

[19]For a partial survey of the literature on determinants of schooling, see Grossman (1975).

[20]The causal effect of high blood pressure in childhood on better oral health in adolescence may also be behavioral. Children with high blood pressure or their parents may have been warned by the examining Cycle II

physicians to modify their diets which resulted in better oral health in adolescence.

[21]If $a(t) = a$ and $b(t) = b$ for all t, and if t approaches infinity, then the cumulative effect is simply $b/(1-a)$. Note that equation (10) is the solved reduced form of our model. It embodies the constraint that, with $h(t-1)$ held constant, g or $h(t-2)$ has no impact on $h(t)$. Therefore, equation (10) differs from the estimated reduced form--the ordinary least squares regression of $h(t)$ on g and e--because the latter does not take account of this constraint.

[22]Although primary and secondary education is financed publicly to a large extent, it is well known that more school resources are allocated to children and adolescents from high-income families.

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Chapter 6

THE CHOICE OF DIET FOR YOUNG CHILDREN AND ITS RELATION TO CHILDREN'S GROWTH

I. Introduction

The purpose of this study is to present empirical results from an ongoing analysis of the household's choice of diet for its young children (0-36 months) and its relation to the children's growth. Our particular interest in this research is the extent to which family income and education of the mother are obstacles to the provision of adequate diets for children in poor American families. The hypothesis that these obstacles are substantial underlies many government nutrition and income support programs.[1]

We have argued elsewhere that the utility of parents is a positive function of their young children's growth[2] and that, subject to genetic, physiological, and wealth constraints, parents influence the growth of their children by their choice of diet for the children and by their investment in their children's health.[3] From this conceptual framework we have derived the following econometric model of children's diet, health, and growth which is consistent with available cross-section data.

$$G = G(D, H, t, z, BW) \quad (1)$$

$$D = D(G, E) \quad (2)$$

$$H = H(D, BW, E) \quad (3)$$

where G, D, and H are endogenous and represent levels of childrens growth (G), nutrient intakes (D), and health (H). Equation (1) is basically a

nutrient intake equations are family income, family size, mother's education, and dummy variables indicating whether the family resides in an urban area, whether the family receives food stamps, and whether the child is or was breast fed.

II. The Data

The data source for this study is the Ten State Nutrition Survey, 1968-70 (TSNS). In this survey, 30,000 families in ten states were selected from low income enumeration districts. Demographic data where obtained from 24,000 of the families by interview. Selected subgroups of infants and young children, adolescents, pregnant or lactating women, and persons over 60 years of age received detailed dietary and biochemical evaluations. Dietary intake data for the previous 24 hours were collected for about 1,700 children less than three years of age by interview of the homemaker. Of these children less than 1,200 could be matched with the characteristics of their family in the editing of the data. This sample was reduced to a working sample of roughly 500 children by deleting all observations (children) with missing data for those variables specified in our model and by deleting all children whose parents reported their age to be 12, 24, or 36 months. This latter procedure was undertaken because of the disproportionate number of responses in these age categories, many of which seemed random in nature. Those observations deleted from the sample by this procedure or because of missing data do not differ significantly from the included observations.[5]

technical relationship, describing how children's growth responds to diet and health levels, given age (t), birth weight (BW), and parental and genetic characteristics (Z). Equations (2) and (3) are primarily behavioral relationships, explaining the choice of diet in the household for the children, given socioeconomic constraints (E), and the subsequent influence of diet on health levels. The rationale for including G in the nutrient intake equation is that larger children demand more nutrients. BW is in the health equation to represent the initial stock of health.

At the empirical level we have estimated several variations of this model of children's diet, health, and growth. The model most consistent with the conceptual framework consisted of seven equations with seven endogenous variables. The endogenous variables were two measures of children's intakes--daily calorie and daily protein consumption; three measures of children's growth--height, weight, and head circumference; and two measures of children's health--the lifetime number of cases of pneumonia and the number of cases of diarrhea in the six months prior to the clinical interview. The health variables, however, did not approach statistical significance in the growth equations, apparently because they had minor or very short-term effects on children's growth that were rapidly overcome.[4] In the presentation of the empirical results, therefore, we emphasize a model of children's diet and growth that has excluded the health variables and health equations. In this model the exogenous variables in the growth equations are measures of genetic and parental traits; namely children's age, sex, birth weight, race and mother's height. Exogenous variables in the

TABLE 1

Summary Statistics

Variable	Mean	Standard Deviation
Daily calories	1,310	653
Daily protein (grams)	55.34	29.80
Weight (kg)	10.56	2.94
Height (cm)	78.26	10.51
Head circumference (cm)	460.95	32.36
Family income	5,353	3,288
Mother's education	10.68	2.75
Family size	5.47	2.23
Race dummy (black = 1)	.27	.44
Food stamp dummy (recipient = 1)	.10	.30
Mother's height (cm)	160.41	6.59
Birth weight (oz)	115.55	20.77
Age	17.80	9.92
Breast fed dummy [child was (is) breast fed = 1)	.25	.43
Sex dummy (male = 1)	.51	.50
Urban dummy (urban location = 1)	.74	.44
Norm height*	.98	.002
Norm weight*	1.00	.007
Norm head*	.99	.002

*See note 7 for definitions of these variables.

Descriptive statistics for those variables included in the model of children's diet and growth are presented in Table 1. Of immediate note are the mean values of the dietary intake and growth variables. Children in this predominantly low income sample had mean calorie intakes of 1,310 and mean protein intakes of 55 grams. The calorie figure is about equal to the recommended dietary allowance (RDA) for children of age 18 months, the average age in our sample. The protein intake figure is more than twice the RDA for children of 18 months. This pattern of average calorie and protein consumption in excess of RDA' remains whether the TSNS data is stratified by age, income, or ethnic group.[6] It is also true that the average of the ratios of height, weight and head growth to the relevant national norms (age and sex specific) average near unity in each case.[7] The ratios of height and head growth to national norms are particularly noteworthy statistics. If we assume that in the U.S. diets and health levels on average are sufficient to insure that height and head growth of children reach the limits of their physiological potential, then the means of unity in the TSNS sample for the height and head growth norms imply that these children are also near their physiological potential.

The finding of adequate or better than adequate protein and calorie intakes among a low income sample of the U.S. population is not an isolated one. The pre-supplement protein and calorie intakes for children 12 to 23 months in the sample drawn to evaluate the Special Supplemental Food Program for Women, Infants and Children (WIC) were 52.9 grams and 1,290 calories respectively, or nearly identical to the figures of TSNS sample (Edozien, et. al. 1976). Average 1973 after-tax

household income was $3,800 for all households participating in the WIC evaluations (41,330 infants and children were in the sample).

Further information on the nutritional status of poor American children can be obtained by examining the diets of children light for their age and sex in low income households. In our TSNS working sample the calorie and protein intakes of children below the 10th percentile in weight for their age and sex were 1,297 and 54.4 grams, or roughly equal to the full working sample mean and indicative of adequate or more than adequate intakes.[8] Unless present and past nutrient intakes are not correlated, these numbers imply that influences other than diet may be responsible for producing the condition usually associated with under-nutrition. The consideration of the structural equation estimates in the next section will enable us to come to firmer conclusions about the role of socioeconomic variables in the choice of diet by parents for their children and about the subsequent effect of nutrient intakes on children's growth.

III. Structural Equation Estimates

A. Protein and Calorie Intakes

The second state estimates of the protein and calorie equations are presented in Table 2.[9] The results are similar for both nutrients. Simply stated, they indicate that children in these low income families get the amount of calories and protein that they "ask for." The child's demand for nutrients (represented by weight of the child) is a very important determinant of intakes.[10] Both family income and mother's education are insignificant determinants of nutrient intakes. These

TABLE 2

Structural Equation Estimates for Protein and Calories, Two-Stage Least Squares

Dependent Variables	Independent Variables			
	Weight	Family Income	Mother's Education	Family Size
Calories	112.3 (10.1)	-.008 (-.8)	-4.11 (-.4)	-30.1 (-2.3)
Protein	4.05 (7.5)	-.002 (-.4)	-.14 (-.3)	-1.67 (-2.7)

^ indicates predicted value.

t statistics in parentheses.

N = 463.

results and the summary statistics showing that the children in our sample have normal rates of growth and development and on average have diets adequate in calories and proteins imply that low income households in the United States have sufficient income and education to provide satisfactory levels of proteins and calories for their children.[11]

The interpretation of the significant family size variable is complex. The results are not consistent with the notion that, family income constant, family size is negatively related to nutrient intakes of children due to a per capita income effect. This is because with family size constant, increases in family income do not lead to increases in nutrient intakes. The negative coefficient on family size could indicate that parents with larger numbers of children are inefficient in the provision of nutrients to their children.[12] In elasticity terms, the family size effects are very small for both nutrients (about -.05 in each case).

B.2 Children's Growth

The second stage estimates of the children's growth equations are presented in Table 3. The protein variable has been excluded from the weight equation because it was statistically insignificant if calories was also included as an explanatory variable. Calories, however, approached statistical significance in these equations even when protein also appeared. The number of calories then seems to better explain weight growth than the protein content of the diet. These results do not indicate that protein is an unimportant determinant of children's weight. Protein and calories are highly colinear ($r = .88$) so a good portion of the protein influence is captured by the calorie variable.

TABLE 3

Structural Equation Estimates for Height, Weight, and Head Circumference, Two-Stage Least Squares

Dependant Variables	Independent Variables							
	Calories	Protein	Age	Age Squared	Sex Dummy	Mother's Height	Birth Weight	Breast Fed
Weight	.002 (1.2)		.36 (4.5)	-.004 (-2.8)	.65 (4.3)	.02 (1.9)	.03 (6.5)	
Height		.013 (.8)	1.54 (10.3)	-.02 (-4.9)	1.92 (5.6)	.14 (5.2)	.05 (6.6)	
Head circumference		-.19 (-.9)	7.4 (9.6)	-.13 (-7.8)	11.4 (7.5)	.35 (2.9)	.13 (3.5)	2.6 (1.4)

^ indicates predicted value.

t - statistics in parentheses.

N = 463.

An argument with a similar framework explains why protein appears in the height and head growth equations while calories does not.[13]

The most striking difference between the results for height and head growth as compared to weight growth is the sensitivity of these variables to nutrient intakes. The elasticity of weight with respect to calories is .26 while the elasticities of height and head circumference with respect to protein are .02.[14] It appears then that, in contrast to weight, the height and head growth of children, given birth weight, proceed on a course determined largely by age and the genetic influence of mother's height. There is little the household decision makers can do to alter these paths if protein intakes are restrained to that range characterizing the TSNS sample. The weight gain of children, while also influenced by birth weight, age, and mother's height, is much more subject to intervention by homemakers via the choice of diet than height and head growth.

Breast-feeding, holding protein intake constant, may have a positive but small impact on head size. This would be consistent with the hypothesis of Jelliffe (1976, p. 1229), who argues that breast feeding may be preferable to bottle feeding because the high lactose and fatty acid content of human as opposed to cow's milk, "seem biochemically designed to facilitate the main characteristic of the new born which is the rapid growth in size and complexity of the brain."[15] The breast feeding dummy was insignificant in the height and weight equations.

IV. Summary and Implications

The empirical results have indicated that low income families in the United States choose calorie and protein intakes for their children that maximize their growth potential, at least as far as height and head size are concerned. The low elasticities of these growth variables with respect to protein and a level of protein consumption substantially in excess of recommended dietary allowances together imply that these low income parents have pushed the growth of their children through choice of diet nearly as much as possible. We have also found mother's education and family income to be insignificant determinants of protein and calorie intakes. Thus, the picture that has emerged is that the education and income levels in low income households are generally sufficient for the provision of adequate intakes of protein and calories for children in the household. This is not to say that the diets of young children in low income households are generally sufficient in all nutrients. Endozien, et al. (1976) have argued, for example, that there are known inadequacies of iron, vitamin A, and vitamic C in low income children in the United States. To the extent this is the case, a prudent public policy may be to disseminate this information more widely. For it would appear that low income households in the United States provide what they consider to be adequate diets for their children. Protein, for example, is widely accepted as an essential nutrient for the healthy development of children. And our results have shown that low income families generally provide protein, a relatively high priced nutrient, in quantities far beyond recommended levels and to an extent where the

marginal impact on the growth of the children is very small. If family incomes in low income households are insufficient for the purchase of what some observers consider to be the "essentials of life," other essentials appear to be sacrificed for the growth and well being of the children.

FOOTNOTES

[1]In 1969, Charles Upton Lowe, the director of the National Institute of Child Health and Development, told Congress: "One out of every three children under 6 years of age are living in homes in which incomes are insufficient to meet the costs of procuring many of the essentials of life, particularly food" (Chase, 1977). For examples of research into the problem of undernutrition in American school and pre-school children in the U.S., see Christakis (1968), Owen (1969), Sims and Morris (1974), and Owen (1974).

[2]Children's growth enters the utility function in a non-linear fashion and excessive rates of growth (e.g. obesity) can be negatively related to parents utility.

[3]See Chernichovsky and Coate (1977).

[4]This is not to say that children's health is not an important determinant of their growth. It is possible that the two measures of health available to us are not highly correlated with health disorders that have greater consequences for children's growth. Omitting health variables from the growth equations will upwardly bias estimates of the impact of diet on growth to the extent that diet also captures the effects of health on children's growth.

[5]Protein and calorie intakes were slightly less for the deleted children and their height, head and weight growth were slightly greater than the children of the working sample when compared to age and sex specific national norms. Mother's education and family income were also similar for the deleted observations.

[6]Health Services and Mental Health Administration, DHEW (HSM) 72-8133 (1972), pp. 12-13.

[7]If N_{ij} is the average height of children in the U.S. of age i and sex j and n_{ij} is the height of a child in our sample of age i and sex j, then the ratio of this child's height to national norms is n_{ij}/N_{ij}. Averaging this ratio across all observations yields the height growth relative to national norms statistics discussed in the text. The national norms were obtained from National Center for Health Statistics (1976).

[8]The mean age of these light for age children is 21 months, so the RDSs for this group average slightly more than those presented in the text for children of 18 months.

[9]Results for the race, food stamp and urban dummy variables are not presented. These variables were statistically insignificant in the structural equations and in the reduced forms.

[10]By not controlling for age in the nutrient intake equations, we are implicitly assuming that the demand for nutrients does not differ between infants of the same weight, but of different age. To allow

for age effects, age and the square of age were entered into the nutrient intake equations. These variables were significant but reduced the t-value of predicted weight to below one in both the protein and calorie equations. Because the age variables were important instruments in generating the predicted values for weight, the age and predicted weight variables are highly correlated and the second stage estimates are difficult to interpret. One reason why there may not be independent age effects is that growth rates for children less than 36 months vary inversely with age while energy expenditures may vary with age. Thus, these age effects could be offsetting. In any case, it is not particularly important for our purposes whether the weight coefficient reported in the text is capturing age effects. The results we wish to emphasize are those for the socioeconomic variables, which are not sensitive to the specification of the age variables in the nutrient intake equations.

[11]Further support for this view is provided by results obtained for the food stamp variable. The food stamp dummy entered into the nutrient intake equation showed no significant effect of food stamps on protein or calorie intakes. This may indicate that the increase in real income resulting from food stamps is devoted to consumption of other goods rather than food; at the least it implies that food stamps do not affect the protein and calorie consumption of young children in recipient families.

[12]Michael (1972) argues that family size variables can be considered as a proxy for the ability to obtain and make use of information and, thus, are inversely related to efficiency. His argument rests on the assumption that with income and education constant, larger families are less knowledgeable about or less proficient in using birth control information. He argues that this may reflect a general inefficiency in acquiring and using many forms of information.

[13]Because the growth equations formed part of a simultaneous system traditional F tests could not be employed to test the individual and joint contributions of the protein and calorie variables. OLS regressions of the growth equations showed that the calorie and protein variables did not make a significant incremental contribution to explaining the variance in the dependent variables when added jointly to regressions containing the other independent variables. When the diet variables were added individually calories made a significant incremental contribution to explaining weight variance but protein was an insignificant addition to the height and head size equations.

[14]Elasticities computed at the means given nutrient intake point estimates. It should be pointed out that the calorie coefficient approaches statistical significance while the protein coefficients do not.

[15]Jelliffe (1976), p. 1229.

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Chapter 7

AN ECONOMIC ANALYSIS OF THE DIET, GROWTH, AND HEALTH OF YOUNG CHILDREN IN THE UNITED STATES

"One out of every three children under six years of age are living in homes in which incomes are insufficient to meet the costs of procuring many of the essentials of life, particularly food." Congressional testimony of Charles Upton Lowe, Director of the National Institute of Child Health and Development, 1969 (Chase, 1977).

I. Introduction

Interest in the nutritional status of young American children has heightened considerably in the past decade. Much of the concern has resulted from research suggesting varying degrees of under-nutrition in low income American school[1] and pre-school children and from evidence indicating a positive association between children's growth and their intellectual development.[2] In this study we analyze the choice of diet for children one to five years in the United States and its relation to the children's growth and health. We are particularly interested in the extent to which family income and education may be obstacles to the provision of adequate diets for children in American families. The hypothesis that these obstacles are substantial underlies many government nutrition and income support programs and has led to the Congressional mandate of two separate comprehensive national nutrition surveys, The Ten State Nutrition Survey, 1968-1970, and the Health and Nutrition Examination Survey, 1971-1975.

In the previous chapter we used the Ten State Nutrition Survey (TSNS) data to examine the nutritional status of children up to the age of 36 months in poor American families. The picture that emerged from the analysis of TSNS data was generally contrary to the impressions left by much previous research. The data indicated that low income parents had pushed the growth of their children through choice of diet nearly as much as

possible. Protein, a relatively high priced nutrient, was consumed in quantities two to three times recommended dietary standards and to an extent where its marginal impact on the growth of children was very small. Family income and mother's education were shown not to be significant (in the statistical sense) barriers to the provision of adequate protein and calorie intakes for children in poor American families. In the TSNS data average protein and calorie consumption was in excess of dietary standards whether the data was stratified by children's age, family income, or ethnic group. Protein intakes in these cross-tabulations were consistently two to three hundred percent of dietary standards.

In this chapter we analyze the Health and Nutrition Examination Survey (HANES) data to provide further evidence on the choice of diet for young American children and its effect on their growth and health. This chapter is divided into five sections. In the following section we describe the conceptual framework and specify a general model of children's diet, health, and growth. This is followed by a discussion of the data that includes important descriptive statistics. In Section IV, we present the estimated econometric model. The final section summarizes the research.

II. Concentual Framework

As a point of departure we postulate that the utility of parents is a positive function of their children's growth. That is, within the bounds of perceived norms, parents desire heavier and taller children.[3] For our analysis it is not necessary that this desire be based on known correlations between current period height and weight of children and their current and future period health status and intellectual development. Rather, we only argue that this desire does exist and that parents make sacrifices or forego other pleasures in order to augment the growth of their children.[4]

Although constrained by genetic and physiological factors, parents influence the growth of their children by their choice of diet for the children and by their investment in their children's health (medical care, parental care, sanitary conditions, etc.). The interdependencies among children's growth, children's health and their diet are formalized in the following model.

We begin by relating the parent's choice of the initial diet, D_o, for a new born to birth weight, BW, which is a proxy for the infant's demand for food, and initial period socioeconomic influences, E_o, that impact on the quantity and quality of diet.

$$D_o = f^o(BW, E_o). \tag{1}$$

In each subsequent period the child's growth, G_t, is determined by genetic and parental traits, Z, and by diet, D_{t-1}, and health status, H_{t-1}, in the preceding period. Health status can be interpreted as an efficiency parameter that affects the rate at which nutrients are converted into children's growth. Formally,

$$G_t = f\ (Z,\ D_{t-1},\ H_{t-1}). \tag{2}$$

The diet in each period is a function of the child's growth, which serves as a proxy for appetite or the child's demand for food, and the economic status of the household.

$$D_t = g(G_t,\ E_t). \tag{3}$$

The child's health status is a function of his diet, growth and other inputs which produce good health, X_t,

$$H_t = h(X_t,\ G_t,\ D_t). \tag{4}$$

The levels of X_t are determined by socioeconomic status:

$$X_t = e(E_t). \tag{5}$$

In order to statistically identify certain key relationships and to make the model consistent with available cross-section data, several assumptions are necessary, some of which are explicit in equations 1-5. First, birth weight is considered exogenous to our model of children's growth, diet and health. A more sophisticated model could include birth weight as an endogenous variable and relate it to parental characteristics, diet of the mother and socioeconomic variables. We also assume that some variables are serially correlated (e.g., diet, household income) or constant (e.g., mother's education, parental traits) over t and that the time increments are infinitesimal.

To isolate the role of diet as a bridge from socioeconomic status to children's growth, we can, given the assumptions detailed above, derive the following simultaneous equations from (2), (3), and (4):

$$G = g(\hat{D}, \hat{H}, t, Z, BW) \tag{6}$$

$$D = f(\hat{G}, E) \tag{7}$$

$$H = h(\hat{D}, \hat{G}, E) \tag{8}$$

which specifies D, G, and H as endogenous variables. Equation (6) is basically a technical relationship, describing how children's growth responds to diet and health levels, given age, birth weight and parental and genetic characteristics. Equations (7) and (8) are primarily behavioral relationships, explaining the choice of diet in the household for the children, given socioeconomic constraints, and the subsequent influence of diet and growth on health levels.

III. The Data

HANES is a national sample of the population of the United States, with oversampling of low income families. The entire HANES sample, which was collected between 1971 and 1975 by the National Center for Health Statistics, contains approximately 28,000 individuals between the ages of 1 and 74. Slightly less than 3,000 children aged 1 to 5 years were included in the sample. Dietary intake data for the previous 24 hours were collected for children less than five years of age by interview of the homemaker. A working sample of 2515 was created by deleting all observations (children) with missing data. The roughly 450 children deleted from the sample did not differ significantly from the working sample in terms of age and sex specific nutrient intakes or height, head, and weight growth. HANES is described in detail by the National Center for Health Statistics (1973, 1977).

Descriptive statistics for variables collected in HANES relevant to our analysis are presented in Table 1. Endogenous variables in our econometric specifications are selected from the measures of children's diet, health, and growth. Measures of children's growth are height, weight, and head circumference. Measures of children's health are lifetime number of overnight hospitalizations and number of colds in the six months prior to the medical history. Children's diet is measured by calorie, protein, calcium, iron, vitamin A and vitamin C intakes.

Exogenous variables in the growth equations are measures of genetic and parental traits, namely children's age, sex, birth weight, birth order, race, mother's height and weight, and father's height. Exogenous variables in the nutrient intake and health equations are family income, family size, and dummy variables representing education of the household head and whether the head is female.

Table 1

Summary Statistics

Variable	Mean	Standard Deviation
Daily calories	1516	584
Daily protein (gm)	55.84	23.06
Daily Vitamin C (mg)	78.45	86.73
Daily iron (mg)	8.01	4.51
Daily calcium (mg)	872	469
Daily Vitamin A (IU)	3576	3743
Weight (kg)	15.48	3.82
Height (cm)	97.86	11.95
Head circumference (cm)	49.23	2.21
Hospitalizations	.30	.45
Colds (last six months)	1.42	1.32
Age (months)	42.63	17.62
Sex (1 = male)	.51	.50
Birth weight (oz.)	115.94	19.80
Birth order	1.45	2.19
Race (1 = non-white)	.23	.42
Mother's height (in.)	64.05	2.69
Father's height (in.)	69.87	3.18
Mother's weight (lbs.)	139.14	29.21
Household income	9280	5563
Household size	5.05	2.03
Years of schooling of household head		
Schooling 1 (1 = less than 12)	.37	.46
Schooling 2 (1 = 12)	.38	.48
Schooling 3 (1 = 13 to 16)	.19	.39
Schooling 4 (1 = more than 16)	.06	.24
Sex of household head (1 = female)	.16	.36

The mean family income of $9,280 is considerably below the 1972 national average of $12,500 and is indicative of the oversampling of low income families. The mean calorie and protein intakes of 1516 and 56 grams are considerably above the protein and calorie standards of roughly 1330 and 26 grams for children of the age and weight corresponding to the sample means.[5] This finding of higher average protein and calorie intakes than dietary standards is not surprising given the similar results from the TSNS, a sample characterized by significantly lower family incomes. Children's intakes of calcium, vitamin A and vitamin C average two to three times recommended dietary standards in the HANES working sample. In the case of iron, the average intake is two-thirds of dietary standards.

In column 1 of Table 2, levels of growth, health, and nutrient intakes are presented for children in households falling into the upper and lower thirty percentiles of the poverty index (PIR) distribution.[6] There are no significant differences in height, weight, or head growth between these groups, nor in protein, calorie, vitamin A, or iron intakes. In the cases of vitamin C and calcium intakes a statistically significant difference emerges in favor of the higher PIR group. For both groups mean nutrient intake levels consistently exceed dietary standards, with the exception of iron. There are also no significant differences in hospitalizations although the lower PIR group had a significantly greater number of colds in the six months prior to the medical history. The average family income and household size for the higher PIR group are $14,766 and 4.2. The same figures for the lower PIR group are $3,673 and 5.7.

In the remaining portion of Table 2 similar high and low PIR comparisons are made for blacks and for whites in the working sample. The patterns of statistical significance within these stratifications are similar to that for the sample as a whole.

Table 2

Mean Levels of Growth, Nutrient Intakes, and Health for Families in the Upper and Lower Thirty Percentiles of the Poverty Index Distribution[a]

	All Families			White Families			Non-White Families		
	Lower Thirty Percentile	Upper Thirty Percentile	t[b]	Lower Thirty Percentile	Upper Thirty Percentile	t[b]	Lower Thirty Percentile	Upper Thirty Percentile	t[b]
Daily calories	1498	1510	.45	1520	1514	-.19	1475	1469	-.10
Daily protein (gm)	54.96	56.14	1.08	56.46	56.26	-.15	53.44	55.00	.55
Daily Vitamin C (mg)	69.12	87.43	5.18	67.64	87.94	4.64	70.62	82.56	1.33
Daily iron (mg)	7.89	8.23	1.56	7.97	8.27	1.01	7.80	7.86	.15
Daily calcium (mg)	819	922	4.82	910	935	.93	72.54	79.67	1.33
Daily Vitamin A (IU)	3577	3694	.42	3621	3595	-.16	3532	4177	1.08
Weight (kg)	15.42	15.51	.53	15.41	15.50	.43	15.42	15.54	.27
Height (cm)	97.63	97.94	.57	97.14	97.99	1.31	98.14	97.51	-.43
Head (cm)	49.17	49.24	.80	49.09	49.24	1.21	49.24	49.30	.24
Hospitalizations	.31	.29	-.72	.32	.30	-.91	.29	.24	-1.03
Colds (last six months)	1.51	1.31	-3.46	1.46	1.29	-2.43	1.57	1.54	-.20

[a]The sample sizes for the lower and upper thirty percentiles are, for the entire sample, 1036 and 918; for whites, 521 and 831; for non-whites 515 and 87. The imbalance in the white and non-white categories results from the use of the entire sample thirty percentile cut-off values for these sub-sample stratifications.

[b]t values are for significance test of difference between means.

Further information on the nutritional status of young American children can be obtained by examining the diets of children light for their age and sex. In our HANES working sample the calorie and protein intakes of children below the 10th percentile in weight for their age and sex are 1440 and 53 grams, not significantly different from the working sample means and indicative of more than adequate intakes of these nutrients according to dietary standards. The mean family income for this group of light children is $8470. Unless present and past nutrient intakes are not correlated, these numbers imply that influences other than diet may be responsible for producing the condition usually associated with undernutrition. The consideration of the emprical results in the next section will enable us to come to firmer conclusions about the role of socioeconomic variables in the choice of diet by parents for their children and about the subsequent effect of nutrient intakes on children's growth.

IV. Empirical Results

At the empirical level we have estimated several variations of our model of children's diet, health, and growth. With the exception of calories and protein, the nutrient intake variables did not approach statistical significance on the growth equations, either because of their high correlations with protein and calorie intakes or because they have very small impacts on growth at the margin. The health variables also performed poorly in the growth equations in the statistical sense, apparently because these conditions have minor or very short term growth effects that are rapidly overcome.

In the presentation of the empirical results, therefore, we emphasize a model with the following endogenous variables: height, weight, head circumference, protein intake, and calorie intake. We also report results for the colds variable and for vitamin C intake.

A. Reduced Forms

The reduced form relationships derived from equations (6)-(8) relate children's growth, health, and nutrient intakes to genetic and parental traits measured by age, sex, race, parent's heights, mother's weight, birthweight and birth order; and socioeconomic influences measured by household income, household size, and dummy variables indicating the education of the household head, and whether the head is female. The reduced form results are presented in Table 3. We are particularly interested in the children's growth reduced forms because of the information they provide on the significance of the genetic and parental trait variables versus the socioeconomic and behavioral indicators in the determination of children's growth. The results show that the latter set of variables are of limited significance in explaining children's growth. The family income and mother's education coefficients generally have low t-values and the addition of these variables and household size to children's growth regressions that already include the genetic and parental trait variables only slightly reduces the unexplained variance in the dependent variables.[7]

Another interesting aspect of the reduced form results is the low R^2's of the nutrient intake equations, none of which exceed .14. The fact that the exogenous variables in our model explain such a small proportion of the variation in nutrient intakes brings to question the importance of these variables in the diet decision for young children.

B. Children's Growth

The simultaneous equation estimates of the children's growth equations are presented in Table 4.[8] The protein variable has been excluded from the weight equation because it was statistically insignificant if calories were also included as an explanatory variable. Calories, however, approached statistical significance in these equations even when protein also appeared. The number of calories then seems to better explain weight growth than the protein content

Table 3

Reduced Form Estimates[a]

Independent Variables	Dependent Variables						
	Weight	Head Circumference	Height	Calories	Protein	Vitamin C	Colds
Constant	4.71	36.21	20.73	-278.48	6.06	17.76	2.81
	(3.59)	(34.61)	(8.45)	(-0.79)	(0.42)	(0.32)	(3.36)
Age	0.14	0.13	0.84	18.55	0.16	1.39	-0.01
	(11.16)	(14.27)	(34.20)	(5.24)	(1.13)	(2.47)	(-1.88)
Age squared	0.000	-0.000	-2.49	-0.09	0.001	-0.01	0.000
	(2.38)	(-7.90)	(-8.84)	(-2.45)	(0.75)	(-1.88)	(0.94)
Sex	0.42	1.12	0.81	149.05	4.17	9.09	-0.02
	(5.21)	(17.14)	(5.32)	(6.40)	(4.66)	(2.61)	(-0.43)
Birth weight	0.02	0.01	0.45	0.04	0.01	-0.03	0.001
	(12.53)	(9.18)	(10.12)	(0.07)	(0.65)	(-0.34)	(0.90)
Birth order	-0.05	-0.009	-0.08	-1.37	-0.16	-0.99	0.001
	(-1.92)	(-0.42)	(-1.55)	(-0.18)	(-0.56)	(-0.85)	(0.06)
Mother's weight	0.01	0.001	0.70	0.64	0.28	0.05	-0.001
	(5.95)	(1.13)	(2.41)	(1.55)	(1.67)	(0.79)	(-1.09)
Mother's height	0.07	0.05	0.37	8.38	0.09	-0.37	-0.001
	(4.54)	(4.32)	(12.15)	(1.91)	(0.51)	(-0.53)	(-0.16)
Father's height	0.05	0.03	0.24	6.72	0.30	0.39	-0.002
	(4.37)	(3.20)	(9.98)	(1.89)	(2.12)	(0.70)	(0.32)
Race	0.40	0.35	1.41	48.31	-2.61	0.42	0.29
	(3.45)	(3.82)	(6.42)	(1.54)	(-2.04)	(0.85)	(3.92)
Income	0.000	0.000	0.000	0.000	0.000	0.000	-0.000
	(2.15)	(1.77)	(2.77)	(0.13)	(0.60)	(2.37)	(-1.51)
Household size	-0.04	0.002	-0.10	18.77	0.80	-0.53	-0.02
	(-1.33)	(0.11)	(-1.93)	(2.32)	(2.43)	(-4.21)	(-1.41)
Schooling 2	-0.01	0.005	0.15	113.30	3.79	5.19	-0.12
	(-0.11)	(0.06)	(0.81)	(4.25)	(3.49)	(1.23)	(-1.94)
Schooling 3	-0.01	0.16	0.34	81.40	3.73	13.92	-0.05
	(-0.08)	(1.65)	(1.45)	(2.39)	(2.69)	(2.58)	(-0.69)
Schooling 4	-0.63	0.05	-1.40	56.97	3.78	9.22	0.08
	(-3.26)	(0.36)	(-3.84)	(1.09)	(1.78)	(1.11)	(0.66)
Sex of head	-0.18	0.09	-0.30	125.51	5.55	-1.44	-0.05
	(-0.14)	(0.95)	(-1.28)	(3.69)	(4.00)	(-2.67)	(-0.68)
$\bar{R}^2$	.71	.46	.89	.14	.08	.02	.03
F	278.9	96.0	973.0	18.8	10.5	3.1	5.2
N	2515	2515	2515	2515	2515	2515	2515

[a] t statistics in parentheses.

Table 4a

Structural Equation Estimates for Children's Growth and Health, Three Stage Least Squares[a]

Independent Variables[b]	Dependent Variables			
	Height	Weight	Head Circumference	Colds
$\hat{\text{Protein}}$	.087 (4.39)		.22 (11.76)	.01 (1.49)
$\hat{\text{Calories}}$		.002 (5.02)		
$\hat{\text{Vitamin C}}$				.011 (.10)
Age	.84 (31.52)	.09 (5.71)	.24 (8.57)	-.17 (-8.6)
Age squared	-.002 (-9.45)	.001 (5.10)	-.002 (-7.10)	.001 (6.91)
Sex	.48 (2.63)	.13 (1.28)	.36 (1.84)	-.82 (-6.11)
Birth weight	.003 (8.53)	.02 (11.06)	.002 (.72)	
Birth order	-.19 (-3.82)	-.07 (-4.21)	.10 (3.36)	
Mother's height	.51 (20.8)	-.11 (-.92)	.42 (23.96)	
Father's height	.40 (18.11)	-.06 (6.13)		
Mother's weight	.002 (1.03)	-.008 (5.61)	-.01 (-7.3)	
Race	1.24 (5.19)	3.1 (2.53)	-.71 (-3.24)	
Income				-.001 (-6.44)
Household size				.06 (1.86)
Schooling 2				.72 (-4.90)
Schooling 3				-1.40 (-6.83)
Schooling 4				-.79 (-2.84)

[a] N = 2515

[b] $\hat{\ }$ indicates endogenous variable.

TABLE 4b

Elasticities of Selected Variables[a]

Independent Variables[b]	Dependent Variables		
	Height	Weight	Head Circumference
$\hat{}$ Protein	.05		.25
$\hat{}$ Calories		.20	
Age	.38	.42	.21
Birth weight	.004	.16	.005
Mother's weight	.004	.07	.01
Mother's height	.33	.46	.54
Father's height	.27	.27	
Race	.003	.001	-.005
Sex	.003	.004	.003

[a] Computed at mean values of dependent and independent variables.

[b] $\hat{}$ indicates endogenous variable.

of the diet. Protein and calories are highly colinear (r = .82) so a good portion of the protein influence is captured by the calorie variable. An argument with a similar framework explains why protein appears in the height and head growth equations while calories does not.[9]

In elasticity terms the most important variables in the growth equations are children's age and height of the mother and height of the father. These results were expected and demonstrate again the importance of variables beyond the influence of the household decision maker in the children's growth process. A result that is surprising is the rather substantial elasticities of children's growth with respect to nutrient intakes. The elasticities (at the means) of height and head circumference with respect to protein are .05 and .25, respectively, and the elasticity of weight with respect to calories is .20. These results imply that an increase in daily protein consumption of ten percent or about five and one-half grams would increase height by an average of one-fifth of one inch and head circumference by an average of one-half of one inch. A ten percent increase in calorie intakes would increase children's weight by an average of seven-tenths of one pound. The protein elasticities in the height and head circumference equations seem particularly large in light of the fact that protein intakes average more than twice dietary standards. The protein effects on growth seem to be linear throughout the range of intakes characterizing the HANES working sample: that is, we do not appear to be approximating a non-linear protein effect with a very small impact on growth at the margin.[10]

These substantial elasticities of growth with respect to nutrients that are consumed in excess of dietary standards is consistent with the findings of the evaluation of the Special Supplemental Food Program for Women, Infants and Children (WIC). This analysis showed that although children in poor American households generally consumed nutrients well in excess of dietary standards, their growth could be accelerated by increasing nutrient intakes. In light of

these findings the WIC evaluators recommended a reassessment of dietary standards and singled out protein in particular (Edozien, et.al., 1976).

C. Nutrient Intake Equations

The simultaneous equation estimates of the protein, calorie, and vitamin C equations are presented in Table 5. The results are similar for each of the nutrients. Simply stated they indicate, within the context of our model, that children get the amount of these nutrients that they "ask for." The child's demand for nutrients, represented by weight of the child, is a very important determinant of intakes. The nutrient-weight elasticities are about one in each case and the t-values of the weight coefficients are substantial. The family income coefficients approach statistical significance but imply very small elasticities (about .02 in each case).

Education of the household head has a positive but nonlinear effect on nutrient intakes. Children in families where the head has 12 years of schooling receive about five percent more of these nutrients relative to children in families where the head has less than 12 years of schooling. However, this education differential falls when children in families where the head has college or graduate education are compared to children in families where the head has less than 12 years of schooling.

V. Summary

A primary purpose of this study was to investigate the extent to which family income and education are obstacles to the provision of adequate diets for young children in the United States. Based on our examination of the HANES data we have found that:

1. Average nutrient intakes of young children are well above recommended dietary standards, with the exception of iron.

TABLE 5a

Structural Equation Estimates for Children's Nutrient Intakes[a]

Independent Variables[b]	Dependent Variables		
	Protein	Calories	Vitamin C
Weight	4.03 (12.78)	78.80 (10.36)	.89 (.93)
Age	-.18 (-1.06)	11.43 (2.80)	2.24 (4.27)
Age squared	-.003 (-1.9)	-.02 (-5.07)	-.002 (-4.89)
Sex	2.25 (2.38)	111.1 (4.89)	9.30 (2.65)
Income	.001 (1.92)	.003 (1.46)	.001 (3.03)
Household size	.60 (3.32)	14.27 (3.13)	-.95 (-1.12)
Schooling 2	2.32 (2.68)	82.1 (3.71)	6.01 (1.47)
Schooling 3	2.39 (2.28)	52.6 (1.94)	15.35 (2.93)
Schooling 4	.09 (.05)	-23.1 (-.56)	9.71 (1.21)
Sex of head	-.02 (-.03)	17.72 (.70)	-2.87 (-1.16)

[a] $N = 2515$.

[b] ^ indicates endogenous variable.

Table 5b

Elasticities of Selected Variables[a]

Independent Variables[b]	Dependent Variables	
	Protein	Calories
Weight	1.11	.80
Age	.31	.32
Sex	.02	.04
Income	.02	.02
Household Size	.05	.05

[a] Computed at mean values of dependent and independent variables.

[b] ^ indicates endogenous variable.

2. Average nutrient intakes for children in households of lower economic status are very similar to intakes of children in households of higher economic status. Rates of children's growth are also similar in these households.

3. Family income and education of the household head have statistically significant but very small positive effects on the nutrient intake levels of young children in the model of children's diet, growth, and health estimated in this paper.

These findings are very consistent with those from a similar analysis we performed with the Ten State Nutrition Survey. A most interesting result of the present study is the rather substantial estimated effects of protein intakes on children's height and head growth, even though protein is consumed well in excess of dietary standards. This finding and the apparent correlation between children's growth and their intellectual development brings to question the adequacy of present protein standards. Could American mothers, who provide very high protein diets for their children in households at all levels of socioeconomic status know more about what constitutes an adequate diet for their children than the experts do?

Footnotes

[1]For examples of research into the problem of undernutrition in American school and pre-school children in the U.S., see Christakis (1968), Owen (1969), Sims and Morris (1974), and Owen (1974).

[2]Owen (1977), in his review of the effects of nutrition on growth and cognitive development, concludes that the "evidence, which still should be considered preliminary in nature, ... [indicates] that bigger is smarter, at least among pre-school children."

[3]More formally, it could be argued that rates of children's growth enter the utility function in a non-linear fashion and that excessive rates of growth (e.g. obesity) are negatively related to parent's utility.

[4]It is often pointed out that in agricultural societies parents are very concerned about the size of their children because physical strength is an important correlate of individual output. Although a desire for larger children in modern societies may not be based on a similar observation, there is evidence that the height or weight of children at younger ages correlate with their intellectual development and health in later years, and thus with their future earnings.

[5]The dietary standards cited in the text are those of the HANES dietary standards committee for children 24-47 months weighing the sample mean of 15.5 kilograms.

[6]As computed in HANES, the poverty index ratio takes account of household income, household size, and household diet requirements as reflected by the age distribution of the household members.

[7]Adjusted R^2's increased by less than .01 when the socioeconomic variables were added to either height, weight, or head size regressions that already contained age, the square of age, sex, parent's height, birthweight and birthorder. It should also be pointed out that the limited significance of the socioeconomic variables does not appear to be due to colinearity with the genetic and parental trait variables. The t-values of the socioeconomic variables do not increase markedly even when the genetic and parental trait variables are excluded from the children's growth equations.

[8]The results for the colds variable are also presented in Table 4 but are not discussed in the text. Household income and education of the household head are inversely related to the number of children's colds while protein and vitamin C intakes do not have statistically significant impacts.

[9]Because the growth equations formed part of a simultaneous system traditional F tests could not be employed to test the individual and joint contributions of the protein and calorie variables. Results from OLS regressions indicate that protein makes a significant incremental contribution to explaining the variance in height and head growth when added to regressions containing the other independent variables, while calories does not. When both diet variables are added jointly to height and head growth regressions the incremental contribution is insignificant. For the weight equation, the incremental contribution to explained variance is significant when the protein and calorie variables are entered individually or jointly to regressions containing the other independent variables.

[10]Predicted protein and the square of predicted protein were entered as independent variables in the height and head circumference equations in the final stage of a two-stage least squares process. No evidence of a non-linear protein effect was uncovered.

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Chapter 8

SUMMARY OF FINDINGS

Our most important findings pertain to the nature-nurture controversy; the impacts of preventive care and health manpower; income and race differences in children's health, the multidimensional nature of children's health and relationships among growth, diet, and family background. These findings are highlighted below.

1. Nature-Nurture Controversy

The home environment in general and mother's schooling in particular play an extremely important role in the determination of children's health. It is not surprising to find that a child's home environment has a positive impact on his health with no other variables held constant. Moreover, it is difficult to sort out the effect of nature from that of nurture because it is difficult to measure a child's genetic endowment and because genetic differences may induce environmental changes. Nevertheless, we have accumulated a number of suggestive pieces of evidence on the true importance of the home environment. With birth weight, mother's age at birth, congenital abnormalities, other proxies for genetic endowment, and family income held constant, parents' schooling has positive and statistically significant effects on many measures of health in childhood and adolescence. Children and teenagers of more educated mothers have better oral health, are less likely to be obese, and less likely to have anemia than children of less educated mothers. Father's schooling

plays a much less important role in the determination of oral health, obesity and anemia than mother's schooling. The latter findings are important because equal effects would be expected if the schooling variables were simply proxies for unmeasured genetic endowments. Moreover, they are especially strong results because oral health, anemia, and obesity reflect health measures that can be modified by inputs of medical care, dental care, proper diet, and parents' time.

Several additional pieces of evidence underline the robustness of the above finding. The relative magnitude of the effect of mother's schooling on oral health, measured by the number of decayed permanent teeth in adolescence, is not altered when the periodontal index, a proxy for genetic oral health endowment, is held constant. When oral health is examined in a longitudinal context, mother's schooling dominates father's schooling in the determination of the periodontal index in adolescence, with the periodontal index in childhood held constant. Similar comments apply to the effect of mother's schooling on school absence due to illness in adolescence (with school absence due to illness in childhood held constant) and to the effect of mother's schooling on obesity in adolescence (with obesity in childhood held constant).

The identification of plausible mechanisms via which mother's schooling influences children's health has increased our confidence that the impact of schooling represents a behavioral effect as opposed to a genetic effect or a statistical artifact. Children of highly educated mothers come from small families and are likely to have seen a dentist for preventive reasons in the past year. In

turn, family size is negatively related to several measures of adolescent health, and preventive care is positively related to oral health. Another mechanism is that cognitive development in childhood has a positive effect on health in adolescence, and cognitive development is positively related to mother's schooling.

To summarize, we have evidence that nurture as opposed to nature plays an important role in the health of children and youths and that the effect of mother's schooling is behavioral as opposed to genetic. Thus public policies aimed at children's health must try to offset the problems encountered by children of mothers with low levels of schooling; in particular, they should try to improve the skills of uneducated mothers in their capacity as the main provider of health care for their children.

2. Impacts of Preventive Care and Health Manpower

With regard to the role of preventive dental care, youths who received a preventive dental check-up within the past year and youths exposed to fluoridated water have much better oral health than other youths. Moreover, the probability of a preventive examination is positively related to the number of dentists per capita in a youth's county of residence. This implies that a program to increase the availability of dentists in medically deprived areas would improve the oral health of youths in these areas. Indeed, we estimate that the payoffs to increasing dental manpower by one per thousand population are about the same as the payoffs to the coverage of preventive dental care under national health insurance.

The probability of obtaining a preventive check-up by a doctor is also positively related to family income and to the number of pediatricians per capita in the county of residence. But we have little evidence that preventive care delivered to youths by physicians is efficacious in terms of their physical health. Therefore, the payoffs to national health insurance for physicians' services delivered to youths or programs to increase the availability of doctors who treat youths are very small.

Overall, what these results suggest is that selective rather than general programs would be most effective in improving the health of the population under 18 years of age. For instance, instead of providing complete coverage for physicians services delivered to persons from birth to age 18 under national health insurance, the government should direct its attention at prenatal care and physicians services during the first year of life. It is known that appropriate prenatal and infant care can make a difference in terms of health outcomes. Conversely, our results for oral health suggest that the payoffs to the coverage of dental care from the age it is first received until age 18 or beyond would be substantial.

3. Income and Race Differences in Children's Health

We show that when health measures from mid-childhood are the subject of analysis, both income and race differences are much less pronounced than they are in infant mortality and birth weight data. We do find differences in the health status of black and white children and of children from high and low income families, but these differences

by no means overwhelmingly favor the white or high-income children. With respect to differences by race, whether or not they are adjusted for differences in associated socioeconomic factors, black children in many cases are in better health than their white counterparts. In the case of income differences in health, the high income children do appear to be in better health according to most measures, but their advantage is greatly diminished when one controls for related socioeconomic factors like parents' educational attainment. These findings imply that policies that aim to improve the well-being of children via income transfers, such as those advocated by the recent Carnegie Council on Children would have, at best, very small effects on health.

4. Multidimensional Nature of Children's Health

Our findings highlight the necessity of explicitly recognizing the multidimensional nature of health. For example, poor and black children are in worse health when traditional health measures are used, but they tend to be in better health when aspects of the new morbidity are under study. Such fine distinctions are hidden when a single index like infant or childhood mortality is used. These results underscore the importance of treating children's health status as multidimensional and illustrate how the use of a single health index could lead to erroneous conclusions about health status and its relation to income and race.

5. Diet, Growth, and Family Background

Average nutrient intakes of infants and young children are well above recommended dietary standards. Moreover, nutrient intakes for children in households of lower economic status are very similar to intakes of children in households of higher economic status. Rates of children's growth are also similar in these households.

There are substantial effects of protein intakes on children's height and head growth, even though protein is consumed in excess of dietary standards. This finding and the apparent correlation between children's growth and their cognitive development brings to question the adequacy of present protein standards. Could American mothers, who provide very high protein diets for their children in households at all levels of socioeconomic status know more about what constitutes an adequate diet for their children than the experts do?

In general family income and parents' schooling have statistically significant but very small positive effects on the nutrient (calories and protein) intakes of young children. Hence, we have little evidence that intakes of calories and proteins are mechanisms via which family background affects the growth of young children. Indeed, when nutrition is not held constant, the impacts of family background on height, weight, and head circumference are very small.